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THE PLACE OF THE PATHOLOGIST IN MODERN MEDICINE.¹

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I REMEMBER hearing with a little alarm tempered by my usual scepticism a distinguished doctor make a remark about pathologists which has turned out not to be true. I had just finished reading my paper on the pathological aspect of cancer at the plenary session on that disease at the Australasian Medical Congress (British Medical Association) at Hobart in 1934. In the discussion which followed, this very distinguished worker in close contact with all phases of cancer work, including physical methods of treatment and research, jocularly said that the "dead-meat men", as presumably I was then, had little further to contribute, at least to cancer, in the way of either treatment or research. The future lay with the physicists and the experimental cytologists, according to the report of this important person. I do not think R. A. Willis had by that time published his classical work "The Spread of Tumours in the Human Body", but it appeared soon after, to be followed many years later by his almost equally erudite "Pathology of Tumours".

This prophecy has not come true: on the contrary, I can hardly think of any branch of medicine in which advances have been made on such an extremely wide front as in clinical pathology.

It is my purpose to discuss tonight how all this knowledge can be applied at the various levels at which the

laboratory is called on to serve the public, from the small isolated hospital unit to the huge buildings, expensively equipped and staffed, for research in private, governmental and commercial foundations.

In the last generation an enormous revolution in laboratory medicine at least has occurred. Those who were able to see the early developments in Great Britain and possibly also in France after the last war could form some idea of the trends and rate of fruition which have given the results today—laboratories the whole work of which is quite beyond the intellectual scope of one individual, however much principles may be simplified. But principle is so often modified by technique and a host of imponderables that, like surgery or medicine, clinical pathology has become compartmented and specialized, to use the common jargon.

I cannot say that I remember Sims Woodhead, the first professor of pathology at the University of Oxford; but I first studied pathology at the University of Sydney under the then best teacher of the whole course. I say this because the professor was the first real whole-time professor of the subject, his predecessor lecturing in some other subject as well as in pathology. I have an idea it was Camac Wilkinson who went to London to try to convince his colleagues there that tuberculosis was curable by injections of tuberculin. Sims Woodhead at Oxford and Greenfield at Edinburgh had had a profound effect on their contemporaries, since in these universities chairs of pathology had been founded. Though British pathology had even then a sound tradition and an honourable history, its pursuit had mainly been in the hands of the great clinicians such as Bright, Addison and Lister, who had been their own pathologists. The German school was really the first to specialize, and what a flowering of genius and talent there was in Virchow, Cohnheim and Rokitsansky! The enormously detailed care and industry

¹ Read at a meeting of the Queensland Branch of the British Medical Association on ??, 1950.

of the German school attracted British and American workers right down to a few years after February, 1933, when steps were taken to dismantle all German science including medical science. Aschoff was the last of the line, but he died without telling us what he thought about it all. His methods and demonstrations at Freiburg-im-Breisgau were the model for teaching all over the world. Nothing like it, I am certain, will be seen in Germany for another generation.

Sims Woodhead must have been alive about the time of the beginning of the first World War, since the water-testing outfit by which pure potable chlorinated water could be offered to all British troops was his discovery. British pathology made its second contribution to the welfare of the nation at war in this way (Wright's antityphoid vaccine was the first). But up to that time the pathologist was regarded as what my colleague at Hobart called "a dead-meat man". Still, the contributions he made to morbid anatomy and histology were of inestimable value. And though bacteriology became, shall I say, fashionable for some few years before the war, the leading pathological journal of the world founded by Sims Woodhead, *The Journal of Pathology and Bacteriology*, still retains its original format and typography and grows fatter with each issue. It is curious that British pathologists have never thought it necessary or advisable to found a separate journal of bacteriology.

Thus the "dead-meat man" still haunts the temples of our great goddess, Medicine. And it was so until about the time of the first World War, when the revolution began. To understand all its implications, we must see how the pathologist fared in the early years of the century. At first a small, rather drab room would be allotted, in which the morbid anatomist examined his material by the naked eye and crude lenses and microscopes. The equipment was extremely simple compared with that of today. In many old practical textbooks are illustrations of holders, quite often nothing much more than a short cylinder of tin, which served to steady material for cutting by ordinary blade razors, the tissue being hardened either by formalin or sometimes by ether as in the old Cathcart instrument. I once succeeded in obtaining a fairly satisfactory section on one of these simple microtomes from material sent direct unfixed from the operating theatre. At least, the section was good enough to make a diagnosis possible. The first microscope I used in pathology had the old telescopic focusing adjustment. In addition to the morbid anatomy descriptions of disease and the cutting of sections in the way indicated, the pathologist was required to know some bacteriology. While morbid anatomy and histology remained rather static by reason of the paucity of technical aids such as we have today, bacteriology with an almost entirely unexplored field developed with startling rapidity—so much so that I have witnessed in my own lifetime this branch of science go through the law of diminishing returns. I have seen the curve of bacteriological discovery and activity soar up and then slowly decline, until it reached its nadir in a sterile phase, in which it seemed unlikely that any new species would be discovered; and as bacteriology was to a great extent taxonomic, and as its taxonomy was neatly built up and had all the creases ironed out, the science offered no attraction to adventurous minds—at least not so much as the exciting new discoveries in blood chemistry, vitamins and hormones. Within the last twenty-five years that has been changed, and the chemical and physico-chemical knowledge obtained by the study of microorganisms has been most remarkable. Though we know we shall most probably not see any new species of much importance, we do know that in the study of living things the bacteriologist has again assumed importance—but to our great regret not before many fine minds were lost to other branches of medicine. Was it the fault of the pathologist who lacked foresight? Or was it just that the personnel prepared to engage in the old-fashioned pathological work were so few, because they were so poorly paid and lacked the encouragement of colleagues who took a slight commercial as well as a humanitarian and scientific interest in their profession? These old workers, many of whom I knew and learnt from, were pushed away into attics and basements and were

"whistled up" now and then for a little help. Up to about 1920, the general attitude to the medical laboratory worker was one of amused contempt sometimes tinged with mystification and fear. It was not uncommon to hear a colleague say "I wonder what you jokers are up to now?", conveying the faint suggestion that sometimes something sinister was cooking in the basement cave of magic. It became the custom to regard the laboratory as a place of last resource which was to be used only to confirm clinical findings; it could never deny, as the clinician could never be wrong—at least *ante mortem*. During my thirty years as a pathologist, I have been called on to try to solve not only clinical problems, but medico-legal problems, to help the Crown to deal adequately with malefactors, industrial problems in an attempt to improve physical conditions of workers in industry, social, nutritional and indeed all kinds of difficulties that the social set-up of our time is bound to produce and which can be dealt with only on the basis of fact-finding and experiment devised to reproduce the relevant circumstances. During this long, active life of medical science in the service of all sorts of interests and of the community, I have seen the common attitudes of my earlier colleagues change most noticeably in respect of laboratory medicine, or if you like, clinical pathology. The vast development of this branch of medicine arose out of the work of the old morbid anatomist, and I shall endeavour to show that in the end, when material has travelled from end to end of the long, long echoing corridors of huge and often luxurious institutes and laboratories, we shall still with all the incidentally acquired information at our disposal, want to know in the end what the tissue changes were. And in attempting to answer that question, I shall feel proud to know that Rokitsansky and Greenfield had not lived in vain. The basements and attics and janitors' smell-holes have evolved into colossal laboratories which seem to imply they can give all the answers. This evolution has been so great that, having seen most of it within my own lifetime, I feel that it has all been something of a revolution; but while there have been huge additions to our power to control the medical environment, the focal point has not moved away from the pathologist and the laboratory of clinical pathology. Thus, for the purpose of this paper, I take the word pathologist to mean one who controls the whole field of diagnostic laboratory medicine. Like many words in medicine which have evolved from the defining of a small, rigidly limited system to another associated with a larger and in many respects a different set of conditions, it is a misnomer, since the word does not cover all it now stands for. The word "pathologist" today has broadened its meaning to indicate a person who directs or controls or practises medical laboratory diagnosis.

In the earlier years of the century the number of individual procedures in a diagnostic laboratory could be written on a postcard; today they would cover pages, and details would bring the summary up to a small pamphlet. There is practically no salt, element, vitamin or hormone which is not detectable in body fluids and tissues, and in ideal conditions there is practically no parasite, bacterium, helminth or toxic agent from drugs or intake of poisons which will not eventually yield to detection by laboratory methods. Gross or fine tissue changes can be studied in great detail from autopsy material, or from biopsy, whether anatomical, or obtained by aspiration or suction, or from exudates or cellular elements in other body fluids. The study of the blood as an organ either directly or by marrow biopsy has widened so much that on this organ alone two-volume text-books are being written. And so on. In disease function in whole or in part is affected, with a consequent alteration in structure or hormonal or secretory output: this alteration can be studied. There is, in effect, hardly a disease or clinical complex or whatever we please to call these major and minor crises of functioning on which the pathologist cannot add relevant comment, often cogent and completely convincing, and always with a degree of accuracy which calculation has shown to be such as to reduce the error and make the finding significant.

The laboratory, then, places in the clinician's hand weapons of considerable power, but it still does not relieve

him of the duty of sound detailed clinical investigation first; indeed, I would say that the very diversity, huge as it is, in possible diagnostic laboratory investigations and their very complexity render a carefully prepared clinical picture based on questioning and use of the common clinical procedures still more necessary and still more detailed. It is not enough any longer to tell me that a patient has goitre, for example. To be of real assistance I must know the patient's age and sex and have an opinion as to what kind of goitre the clinician thinks it may be, and why. If a patient has a disease of the bowel, fifty years ago it might have seemed learned for a clinician to say that the patient had lenteric diarrhoea. Today, when we go into action and wish to get quick results for the simple and desirable purpose of saving life and abolishing pain, the pathologist must not be put in the position of having to suggest a variety of procedures; he should have enough information to allow him to go straight to the point. That is the position at the Brisbane Children's Hospital today, and I doubt if any time now is wasted on laboratory diagnosis by exhaustion, as was the case long ago. The clinician has improved along with the improvement in the services we can offer him.

Therefore good diagnostic facilities in the laboratory cannot fail to have a good clinical influence, and if this is to be of the best, the former must also be as fully equipped as the situation demands, as to space, personnel and plant.

From long experience, and particularly from experience of the period of flooding with work of the department at the Brisbane Hospital, I am now convinced that the diagnostic centre, as I shall continue to call the laboratories section of the hospital, should be the centre of any large modern hospital, and the hospital should be built round it. All other designs based on the old way of pathological life should be scrapped. I suppose that the majority of laboratories of older hospitals throughout the world started in attic or dark basement, certainly in some corner that was not coveted by the stronger tribes of the hospital community, such as surgeons of all kinds. The gentle pathologist, almost ashamed of his existence, submissive to the blows of fortune, always frustrated, because he could not get a grant of £5000, for example, for equipment to perform one test every two years for a certain physician on a very rare and mild disorder, has emerged from his darkness and should be allowed to shed his own light on the whole institution. One of the most difficult and painful things for a human to do is change a habit. Now one habit of the past in hospital design was to forget the laboratory and, when reminded of it at the last minute, put it under a tank-stand.

May I relate two harrowing experiences? I saw the first rough ideas and later some blueprints of the present Brisbane Hospital. Later I shall have something to say of the relation to politicians of this problem of provision of adequate diagnostic centres; but just for the moment let me say that on looking at the plans I immediately laid on them the eye of one destined to try to provide laboratory services. In the plans these were completely missing. That was about 1930 or perhaps earlier. I asked where the diagnostic centre was to be, and was authoritatively told "where it is now"; and it is "where it is now". I was asked when the last war broke out to have a look at the plans for a military hospital. One glance was enough. I did not grow angry, as I felt like doing, nor did I resign the job, since I find that resigning in a difficult situation is a victory for the opposition. I stood by, crushing back my tears, to see the laboratory jabbed away into the usual bit of blank left-over space. I said to the colonel in charge that the plans for the laboratory were a disgrace. They had the wrong orientation to start with, and the place is a furnace in summer and quite unsuitable for sound bacteriological work and is miles from anywhere. Luckily I was never during the war called on to work in the place; but my sympathy goes to those who had to do so.

There must be a complete reorientation of hospital design to bring the diagnostic centre as close as possible

to the whole hospital. Of course I realize that every passenger on a ship cannot have a cabin next door to the captain's, just as I realize that a ship must have two ends at a considerable distance one from the other. But much better planning is possible and, I believe, urgently necessary.

The room of the director should be large and designed for conferences, with adequate equipment for such purposes—microscopes and lamps, X-ray viewing box *et cetera*—as well as an adequate library consisting of the books likely to be of common understanding to pathologist and clinician. Frequent consultation either between one clinician and the pathologist and his staff or with numerous clinicians is mutually beneficial.

In 1948 the orthopaedic department ran across a series of bone tumours of unusual difficulty and in greater than ordinary numbers. I formed the practice then, which is occasionally still used, of deferring my histological diagnosis until there had been a consultation between the orthopaedic surgeon, sometimes a general surgeon, the X-ray expert, my resident medical officer and myself. This plan paid well, with benefit to the patient at least in accurate diagnosis, to the surgeon as he had strong backing for his approach, and certainly to myself, as I began to feel more confidence in a most difficult field. Incidentally, the orthopaedic specialist and myself tentatively suggested a research grant to enable a bright young lad to go over a lot of old material in the way of bone tumours in the X-ray and pathology departments. I am sure we have the material for a good paper, and the opportunity came only as a result of close contact between the various departmental staffs; in this particular situation the morbid histology was the central core of the diagnostic problem, which, however, could be solved only by reference to other relevant factors.

In clinical medicine situations must often arise when difficulties of diagnosis or treatment cannot be solved within the purely clinical range and a coordinated planned attack is necessary from many angles. Though much work of this type may merge into research of an *ad hoc* type—a subject to which I shall refer in greater detail later—the holding of a prior consultation on methods likely to be useful and a later one to evaluate and interpret results seems to me a desirable reform. But clearly such a reform is in practice impossible when the diagnostic centre is far away from the main mass of wards. This has been the supreme difficulty for many years in administering the pathology department of the Brisbane Hospital. I have told how I saw the original hospital plan and how I deplored the general idea and feared the consequences in later years. I want to return to that subject later, as mistakes have been made in the past and we should learn from them. But it appears that all we learn from history is not to learn from history. So far as the Brisbane Hospital and associated institutions are concerned, it must be remembered that the present department grew out of a small laboratory founded for the express and sole purpose of examining diphtheria swabs. That needed only one room. The Brisbane Hospital possessed a corrugated iron shed, which in my time was the carpenter's shop and which had been used as a laboratory spasmodically by an occasional resident medical officer in the years before the 1914 war. It possessed a small creaky hand centrifuge, some test tubes of dried media and a clogged haemocytometer, and some superb ash timber in the benches. This latter I transferred to the present department as the only equipment worth while in the place. About 1923, while I was still honorary pathologist to the Mater Misericordiae Hospital, South Brisbane, I was approached by the superintendent of the Brisbane Children's Hospital to see whether it would be possible to extend the usefulness of the little room, as usual tucked in under the main frame of the hospital buildings. The outlook seemed bleak, as anybody who remembers the old building will agree. All the space we appeared to have any chance of enclosing was that between the piers of the building. So we raised the level of the floor from the primordial dirt to the present concrete flooring. At that time, too, the hospital, to the best of my

recollection, was controlled by a committee administering funds contributed by the charitable public, with, I suppose, some sort of government subsidy. Funds at any rate were very short, and the only way to get the equipment we wanted then (and how exiguous it all seems now!) was to buy it ourselves. Not long after, we were doing most of the important branches of serological diagnosis and cutting sections on a real rotary microtome. But it must quite clearly be understood that the siting of the department depended on the fact that the department was to serve the Children's Hospital exclusively. In the meantime the pathological work of the Brisbane Hospital was still being sent two or three miles to the Health Department's Department of Microbiology in Hope Street, South Brisbane. The ground floor is now, I think, used as the department's venereal diseases out-patient clinic. For much, indeed I think most, of the time the microbiology laboratory had no medical head; it was run for a time by a veterinarian who had been a laboratory boy in the laboratory at King's College, London, not long after Lister had left there; in that laboratory I was later to do my own post-graduate study. Doctors came and went from the laboratory, while a particularly fine type of devoted, skilful and sensible laboratory assistant ran the whole show. Of course conditions in the department's laboratory have improved enormously. It is apparent from a perusal of the files that there was some extraordinarily strong and, to me, inexplicable opposition to the establishment of a laboratory at the Brisbane Hospital; finally, in the view of one of my deeply respected colleagues, who was assistant superintendent at the Brisbane Hospital at the time, there seemed to be no alternative but to make use of whatever facilities the Children's Hospital might agree to afford. And thus the laboratory that was originally designed to serve a hospital of a hundred beds or so and a few thousand out-patients a year, by Procrustean necessity had to serve about 2000 beds and a quarter of a million out-patients yearly. This I should say would be typical of what was happening all over the world. And when our government, acting perfectly within its democratic rights, decided to give the public free laboratory and X-ray diagnosis, the appalling flood of work which descended on us so swamped our resources that any attempt to keep abreast of modern developments had to give way to an attempt to survive. I am sure that all old hospitals which had not originally planned for proper expansion must have had a similar experience. Even if these old laboratories had had the space, there was still the eternal problem of adequate staffing. To any worker in this field who had known the subject in the early twenties, the speed of advance in all fields was breath-taking, and of this I shall give one or two striking examples later. Now staffing and size are interdependent, and both obviously depend on the size of the service required calculated in in-patient beds, out-patient population and private work referred by outside practitioners. Owing to the decision to centralize the main services to the population of Queensland between the Maroochy and Tweed rivers, east of the range to the sea, but excluding Ipswich, the pathologist's burden in respect of the hospital population alone is tremendous. Both the principal authors of the idea of centralization are now dead; but as I knew both well, and indeed intimately, I had long discussions with one in particular about the validity or feasibility of the idea. The only hospital systems I knew well were those of London, Sydney and Melbourne, and it seemed to me then—and I still believe—that the decentralized, relatively small hospital serving a limited neighbourhood, but of course sufficiently elastic to take in persons in need of some special service offered by the neighbourhood hospital, such as thoracic, cardiac or cranial surgery of an admittedly superior type, had proved by experience the best idea, and that no new evidence had been advanced to disprove this contention. In 1938 I was asked to submit estimates of floor space of a new department; but the outbreak of war in 1939 wiped out any possibility of improvement of the position until the "cessation of hostilities". I would say, on the evidence, that they have not ceased yet.

During the war the Minister for Health and Home Affairs constituted a committee to advise him on the

foundation of the research body since founded as the Queensland Institute of Medical Research. Amongst other things, it was agreed that there should be free interchange of ideas and help between the institute, the Faculty of Medicine and the Brisbane Hospital pathology department, the whole to be housed in one five-story or six-story block. Even as things are, the mutual help idea has worked well; the institute tackled for us certain work of a research type in bacteriology, and we supplied autopsy material to the institute, which in a series of papers has advanced our knowledge of salmonellosis and has helped greatly to reduce the terrible mortality in the early stages of the epidemic.

This brings me to the question of the relationship of the diagnostic centre to research.

In principle, my experience tends to make me believe that routine work on such a huge scale as that for which I am responsible does not mix well with research. There are some clinico-pathological investigations which could be regarded as perhaps elementary research. I have personally tried to do both and am convinced that I could not do either really well at the same time. The day of the alluvial miner in clinical pathology collecting large nuggets lying around to be picked up by anybody patient and observant enough to look around a little, the day when the curve of bacteriological discovery was almost vertical, is over. Research is necessary, and comes well within the scope of the life duty of every medical man and every hospital, which is threefold—to attend to the sick, to teach and to extend knowledge, if only by case reports of instructive and interesting material.

I believe the ideal set-up to be the type we advised the Minister to adopt here—that is, the research institute, the hospital diagnostic centre and a laboratory at the disposal of the professors of physiology and pathology, all together in the same building. Medicine and all branches of it, particularly the medical sciences, will become static and die without the constant fructification of new ideas and new techniques, along with the constant overhaul of contemporary medical thought and the rectification or amplification of the type we see in the new editions of the pathological text-books. Now it is supererogatory for me to tell a medical audience that this is all very costly. In reply to that, I would say that all sound work carefully done must inevitably pay. In every institution in the world, from the humble cottage to the Rockefeller Foundation, there is always the washing-up to be done; somebody must look after the laundry. That is to say, if you want to investigate some aspect of pancreatic function, you may have to make a tedious series of blood-sugar estimations, which I think should be handed over to the routine diagnostic centre rather than waste the time of persons whose work is more valuable if devoted to other than routine tasks. Similarly, if in the course of routine investigations some interesting question arises relevant either to special issues or to fundamental principles (and I know by experience that such questions are always arising, but remain to us just the ghosts of a question mark), the interesting and promising side-issues should be referred to the research people for a report and possible investigation. Such a plan operates in the Royal Melbourne Hospital, which does all types of research within its walls, and includes the Walter and Eliza Hall Institute of Medical Research—pure research, *ad hoc* investigations and clinical research by a team of highly qualified personnel, which has already turned out some admirable work.

But to me this fructifying influence is much too small in Australia, and it may be interesting to speculate on the reason.

I have nothing but respect for the loftiness of aim, purity of motive and legislative ability of the men and women whom we as a democratic people have voted into our various parliaments and councils to legislate and care generally for the welfare of the nation. Perhaps I am too chauvinist if I look beyond Australia for faults. But to anybody who has examined the political history of, say, Europe including Great Britain, no other conclusion is possible but that statesmanship is continually going bankrupt. In fact the system in France, for example, is a martyr to bankruptcy; international morality, over such

a short period as that between 1920 and 1939, was hopelessly corrupted, lying and treachery being openly and brazenly practised. This allowed into power one of the most remarkable governments the world has ever seen, that in Germany from 1933 to the end of the late war. It was remarkable because it consisted of known criminals and gangsters, led by one of the worst criminals the world has seen, Hitler, who reached the extreme limit in the stigmata of the politician regarded as a biological type. This kind of people's representative must not only regard it as his duty to serve the State, he must also fanatically believe himself indispensable and make a continual display of power either to impress or to intimidate. He will always of necessity persecute helpless minorities and if possible seek prestige abroad by a cold war on innocent neighbours or a resort to armed force. One of the minorities in Germany was that which included honest scientists of all kinds, men of humane feeling who could not subscribe to the rubbishy racial theories of Hitler and Rosenberg. In quite a short time serious science of the best type ceased to exist in Germany, and from the accounts of post-war observers it is not likely that medical science will recover for a whole generation.

In Russia, a most interesting development has occurred. The value of particular scientific theories is gauged by the extent to which they are acceptable to party political leaders. Classical genetics has been wiped out, and little is known of what has happened to medical science beyond what Professor Eric Ashby has told us: he believes that in this sphere up to his time of writing at least work of value was being done. How long this can continue is a matter for speculation when we read of what is happening to writers and composers. It may be possible, though I personally doubt it, to write or compose at a high creative level if the creative mind is, as it were, strait-jacketed. We can only wait for some years to pass to evaluate correctly the efforts of the Russian government to get its writers and musicians into a political groove. That is why I dislike poets-laureate who are likely to be dug out of bed in the middle of the night to write an ode on an earthquake or a train wreck all neat for the morning paper.

Now all this devastation of the mind and work of the intelligentsia has obviously followed the work of our statesmen. I do not wish to persecute this earnest minority of our worthy citizens, nor do I actually blame them, beyond pointing out that in democracies today practically all activity is political and citizens, all of whom must vote, are closely affected in their lives by the actions of their parliamentary representatives. If we look back only at Lord North and Ramsay MacDonald and consider Britain today, we cannot but feel a deep sorrow at the mistakes of the citizen-elect. A great empire is humbled; and it is not the military conquests that I regret, but the fact that the nation with the richest of literary and medical traditions may no longer be able to make the contributions which were unsurpassed and in many ways unequalled in the world. Thinking of the possible damage to our great traditions as I see Britain laid low, I cannot but feel about it as I always do when thinking of the melancholy lines of Samson in Milton's poem:

Promise was that I
Should Israel from Philistian bonds deliver:
Look for that great deliverer now and find him
Eyeless in Gaza at the mill with slaves
Himself a slave under Philistian yoke.

There is all about us a world to be saved, and that can be done only by the application of the intellect and not only of the scientific mind. All creative minds contribute—Einstein writing about relativity, and Gowland Hopkins planning and making his sublimely simple experiment to demonstrate the reality of accessory food factors or vitamins, seem to me to work mentally in a way analogous to Shelley writing "The Revolt of Islam" or "Prometheus Unbound". I choose Shelley because he tried to do what we as medical men want to do—to liberate the human mind from sorrow and the human body from pain.

As I see it, then, in a democracy the creative mind must have unlimited freedom. Looking back on the years from my graduation just before the first World War to the present, through a depression and another World War, I fear that statesmanship may always fail, like the lights going out in a great catastrophe, and that all the work of the mind may perish as it did literally in Germany from 1933 to 1945. I feel that in our own beautiful country, peopled with citizens brought up politically in the best traditions, there is far less to be feared than in any country in the world. I was going to except the United States of America until I read the Press reports of Senator McCarthy's remarks. But I need only point to what has happened here in Australia in the matter of medical research to arouse the ghosts of old anxieties about freedom for the creative mind. Evidently up to the late twenties the scientific atmosphere in this country was uncongenial to many of our greatest intellects. There is no need to repeat the melancholy catalogue of the names of those of our colleagues who have worn a one-way path to Britain and other countries. Here there was a political moral; but it is, I think, still unlearned. Those of us who wanted to see research fostered had hoped to see the best organization established; but I cannot remember what government or what minister decided to set up an organization of the type which was precisely that which Great Britain had tried and rejected. Research is not frill; it is an urgent necessity. While private foundations do make valuable contributions, we must admit that in Australia for the foreseeable future endowment must come from government sources, and this should be used to the greatest advantage; that can be done only by people closely associated all their lives with and eminent in medical research. And no time can be wasted; advances are so rapid that the effort to 'keep up' is breathtaking, so that plans, schemes, blueprints, should all be elastic enough to allow for rapid expansion.

But even if foresight of this kind is practised, the lack of trained personnel is a matter to which I should like to direct attention. There are two difficulties to overcome—first, schools of training, and secondly, finance. In Queensland, we find little difficulty in making training available at the various levels of laboratory skill and knowledge, from bottle-washer to graduate in medical science. This latter course is the only one of its kind in any Australian university or indeed in any British university. But above that, we find the utmost difficulty in obtaining well trained and experienced medical personnel. Maybe we still have not grown accustomed to thinking of clinical pathology as the vast subject it is, and of the medical worker in this field as worth very much monetarily. But I suggest that hospital budgets should make more liberal allowance for medical graduates to stay on in financial comfort to direct the work of the diagnostic centres in the big hospitals, and the smaller laboratories I envisage throughout Queensland in local geographical groups. I think our medical science graduates are capable of running a country laboratory in selected centres under a regional medical director, who alone could deal with certain parts of the work (morbid anatomy and histology I particularly have in mind). Now in recent years there has been much competition for trained workers, and naturally the best workers went to the highest bidders. These may seem small complaints; but the utter devastation for days that can follow the departure of a skilful and experienced worker has to be felt and seen to be understood.

I think it time all the governmental and private agencies employing laboratory personnel should agree on a uniform scale of classifications and salary ranges. I do not propose to make all staffs static, but I do think some continuity of a solid core of workers is necessary, and this can be obtained only by uniformity of the terms of employment. Exchange would be desirable between various institutions, and uniformity would encourage that, since each worker on exchange would have a pretty good idea of the type of work and payment he could expect in his new environment.

What of the future? There is a mountain of arrears in all laboratories of any consequence in the world—not, of course, of reports on daily routine tasks, but of

extensions to meet the rapid output of new techniques. We have to provide research institutes, adequately staffed and equipped, and I might add directed, diagnostic centres under experienced medical graduates (preferably well-grounded pathologists of the old dispensation) properly sited in large hospitals, and smaller laboratories in smaller selected hospitals run on some kind of zoned system under a regional director. There are at least fifteen hospitals outside Brisbane and Ipswich which should have laboratories. Of how the clinical work of the hospitals and private practices can go on without them I cannot form an idea, since it is so long since I started professional life in the bush. The only way I could diagnose typhoid fever then was to see if the patient had leucopenia; that helped, but it was not very much. I think that provision of services of this kind would greatly ease the strain on the city base hospitals.

To make some of my main points clearer, I should like to refer to the extraordinarily rapid advance in some fields of medical work.

During the first World War, high explosive was used for the first time on a really grand scale, so that shock and hemorrhage became major problems, not always related, as one could occur without the other. At that time we did actually think of the two as quite separate entities, and studies were started on that basis. To deal with them blood transfusion was used, and I remember watching the new teams going round wards and operating theatres rescuing people from death. And I must say, too, that anti-shock therapy by warmth, posture *et cetera* was often startlingly good if operation was delayed until initial recovery from the shock state. Sir Henry Dale thought he had discovered the cause of shock, and so did the Nobel Prize Committee, as they awarded a Nobel Prize to Dale. However, he did not get so far as he thought. But all now admit that he not only made a discovery of something, but in addition pointed the way for a new line of investigation. This incidentally is the sort of thing that keeps on happening. New truth is always fruitful, as it always opens up new vistas for the investigator. By the time the second World War had broken out, experimental research into the problem had practically solved it, and now relatively simple laboratory tests can be used to detect its appearance.

It was, however, in the closely allied field of blood loss and treatment by transfusion that advance was so rapid and so fruitful. The first blood transfusion I ever saw was given to a German air pilot suffering from shock who had also lost a lot of blood. After the usual resuscitation preliminaries, he received a transfusion of whole citrated blood. Whether his blood had been typed or whether the cross-matching of today was used I am not sure. I rather think neither was done; but the pilot recovered rapidly. This left a deep impression on my mind, and afterwards in London I made inquiries about the whole question. I heard of many incompatible transfusions and the reason for them. In 1919 I saw a demonstration at a London hospital of typing by the use of A and B sera. That further engaged my interest, and not long after I joined the staff of the Brisbane Hospital, a volunteer blood donor panel was started, and by searching around amongst people whose blood had been typed in England, we were able to get a supply of A and B sera. Not long after that, I read Landsteiner's paper on M and N groups in *The Journal of Experimental Medicine*. Not knowing as much then as I do now, in all innocence I tried to produce these types. The technical difficulties were immense, so I wrote to Landsteiner, who very kindly sent me sample sera, which, however, lost all their potency on the way. At the same time he sent me reprints of many of his papers, and I found one of engrossing interest—the first paper he and Wiener wrote on the Rh factor and its significance. This work of Landsteiner's, like that of Mendel and Jansky, lay unnoticed by the profession generally for many years, since as late as 1938 we were still giving jaundiced babies transfusions with maternal blood. The letter to *THE MEDICAL JOURNAL OF AUSTRALIA*, signed by Mr. Noel Henry and myself, pointing out the danger of such a procedure and the facts as revealed by American workers on the problem

of neonatal jaundice, was, I think, the first notice on the subject in the Australian medical Press. From the time when the discovery was made progress was extremely rapid, and the application of genetical mathematical analysis to the subject by such workers as Race, Taylor and Fisher led the last-mentioned to postulate theoretically the eventual number of antigens in the Rh complex; by now every Rh subgroup has been found in accordance with the predictions of Fisher. This bald description fails completely to convey the sense of romance in science which the extraordinary advances in research produce in the initiated. Of course, one has to admit that technical methods had already been laid down, but one cannot fail to be impressed with the fact that the whole subject—and it is a very large one—was cleaned up and the creases were all ironed out in about twenty years or so. Apart from the great clinical importance of the Rh factor, it seems to me that the detection of subgroups in the population brings appreciably nearer the day when ABO, M, N, Rh factor and subgroup typing of every person in the community can be individualized, and paternity testing will cease to be exclusive and will be direct. I think this is very important, and I am also proud to say that my department has contributed some valuable work to the stock of knowledge of the subject. One of my staff, Mr. Henry, is now a worker of international reputation, and so great is the demand for this service that I have three highly trained staff members devoted to this alone. I have suggested to Mr. Henry that I believe the time has now come to make a survey of Rh subgroups in the Australian population, and have suggested that he try to arrange a conference in Melbourne where at least three workers also making valuable contributions are working. I anticipate interesting results if we can make this survey. I hope Dr. Diamond's visit to the Brisbane Congress may further stimulate thought along these lines. Even as late as ten years ago nobody would have dared to predict that sero-haematology would spread out so rapidly on so wide a front. As the work is delicate though not terribly difficult—knowledge is rather more important than technique, I think—only highly paid personnel are in my department allowed to handle the matter.

In the field of pathology, on the other hand, I think there has been one alleged advance which for the moment I regard with a little scepticism, and that is cytological diagnosis, using the term in a broad sense. In all diagnosis, or nearly all, I think tissue changes fundamental; that is why I have always considered morbid anatomy and ante-mortem or post-mortem histology fundamental. Clinicians are attempting to secure material for cytological diagnosis by various means—ordinary surgical biopsy, aspiration biopsy, tissue culture, and the cytological examination of fluids or exfoliative specimens. To the experienced worker, surgical biopsy presents no difficulties in general; aspiration biopsy can often give a diagnosis and I have found it useful occasionally. But at present, I think the claims for pure cytological diagnosis are a little optimistic. This is no reason, of course, why we should discontinue this kind of study—on the contrary, it should be worked right out to truth or oblivion; but I would not consider anybody competent in this field before at least two and preferably three years' unremitting study. This matter will receive earnest consideration at the forthcoming annual meeting of the Australian Association of Clinical Pathologists and some sort of authoritative opinion of Australian pathologists will be made available.¹ But this case, again, does point to the necessity of expensive long-term work. I should like to conclude my store of examples of how the pathologist should fit into modern medicine by reference to tuberculosis and leprosy, both diseases caused by organisms with a peculiar type of resistance to therapeutic agents. There is still much work to be done on both, especially directed to the pathogenesis of the latter. To the former I have given much attention, and feel after the recent announcements in the Press that the Government proposes to do what we all believed it possible to do.

As long ago as 1928, Dr. A. P. Murphy and myself interviewed the then Commissioner of Public Health, as

¹ The discussion was to have been arranged, but, for cogent reasons, had to be abandoned.

we believed the problem was growing rather out of hand, and that a more rational approach to the disease should be made than just mere notification and fumigation. Our efforts failed, as we could get no real support for our plans. In 1935 I was president of the Section of Medicine and National Health at the Melbourne meeting of the Australian and New Zealand Association for the Advancement of Science, and noted and recorded the section's unanimous motion that adequate pensions should be paid to tuberculosis sufferers, as we believed the disease just as much an economic as a medical problem. The plenary session of the Australasian Medical Congress (British Medical Association) also unanimously adopted this resolution. In the meantime, much anatomical research and many surveys had been made. It was found that though the death rate was declining the disease continued to occur, though it had been authoritatively stated that it could be eliminated in a generation. It was simply a matter of detecting the people with active disease, who were the foci from which it spread, and giving them treatment in conditions which removed them from contact with other people while giving their families and themselves the financial support necessary over perhaps two years. There can be many lines of attack, but the first is mass Mantoux testing followed by radiographic examination of the subjects who give positive results and B.C.G. inoculation of those who do not. The whole mechanism of mass diagnosis is so simple that any laboratory can tackle the job. I mention leprosy as it has been so much in the public prints lately. We still need to know much more of its pathogenesis; but in passing it should be stated that within the last ten years, treatment here in Queensland has improved very much and all the conditions of treatment also have much improved.

But the problems to be tackled at all levels of pathological diagnosis are almost infinite, and at last the laboratory has acquired sufficient skill, knowledge and devotion to help enormously in solving them. All the pathologist needs is space, money, trained personnel and close access to his material. The "dead-meat man" is still a person of great value, since he has grown up and knows more than his father did.

FACIAL PARALYSIS DUE TO FRACTURE OF THE BASE OF THE SKULL: SURGICAL THERAPY.

By J. PARKES FINDLAY,
Sydney.

MANY cases of facial paralysis due to fracture of the base of the skull have been reported in medical literature during the last decade. I have searched the available literature for detailed information relative to surgical therapy in these cases, and to date I have found only one article by Carrera (1944), which is specific, and another by Behrman (1949), which describes his surgical treatment for severance of the facial nerve. Therefore, it seemed to me that a detailed specific account of the clinical investigation with radiological data and precise surgical therapy should be available for the guidance of clinicians and surgeons.

I reported a case of this clinical entity in this journal in 1946, and mentioned several cases in my book on facial paralysis (1950a), and in this journal also (1950b) in February, 1950. In all cases the condition was treated by surgical decompression of the facial nerve.

I do not think that a recitation of the symptoms and signs of facial paralysis and of fracture of the base of the skull would be appreciated. It is important to ascertain the time relation of the facial paralysis to the fracture of the base of the skull. The first point is whether the facial paralysis manifested itself immediately after the accident, or whether it developed after forty-eight hours. The latter type of paralysis may occur up to ten days after a fracture of the base of the skull and be a complete partial or complete massive facial paralysis.

The immediate paralysis is due to a laceration or complete severing of the facial nerve in the Fallopian canal. The delayed paralysis is due to strangulation of the nerve in the Fallopian canal by compression following a hæmorrhage or transudate into the canal, or to pinching of the nerve by the fracture passing through the Fallopian canal with displacement of the fractured segment (this is clearly demonstrated in the X-ray photographs in the case presented). Also it must be noted that the blood supply to the nerve may be involved by the fracture. The blood vessels, more especially the *vasa nervorum*, may be entangled in the blood clot or pinched with the facial nerve. The entanglement and pinching were noted at the decompression operation of the facial nerve in the case presented.

Diagnosis.

The diagnosis is dependent on the clinical picture presented, with emphasis on the differentiation of the time onset of the paralysis, and on the radiological films. As the diagnosis in these cases is intimately associated with the clinical investigation, it is my intention to discuss the routine investigation under the heading of diagnosis.

It is advised that the radiological examination of the skull must include three views: (i) a direct lateral view, (ii) a lateral oblique view, (iii) a vertico-submental view.

The lateral oblique view is most important, as it will show the posterior fossa with the posterior and upper surfaces of the mastoid temporal and squamous temporal bones. This view with the lateral view will confirm any fracture involving this region of the skull. (This is demonstrated in the illustrations to the present article.) To obtain this view it is necessary to place the head of the patient in position for a direct lateral view, with the X-ray tube tilted nearly 20° caudad. The vertico-submental view gives an excellent picture of the middle fossa, especially the *tegmen tympani*, and of the superior surfaces of the petrous temporal and occipital bones. The depressed fracture involving the *tegmen tympani*, or roof of the left attic, is well shown in the illustrations to this article.

It is my opinion that when delayed facial paralysis is present a fracture of the base of the skull should be demonstrated by X-ray examination, especially when it is accompanied by other clinical signs and hæmorrhage from the ear.

Further investigation may be possible if the patients are cooperative and in full possession of their faculties. It may be possible to establish the site of the lesion or fracture in which the facial nerve is involved by testing the reaction to taste on the paralysed side. This will exclude the *chorda tympani* nerve, which leaves the facial nerve in the Fallopian canal about one centimetre above the stylo-mastoid foramen. The test for taste includes sweet, bitter, sour and salt flavours, either painted or applied on the side of the protruded tongue by a glass applicator. If any interference with hearing (hyperacusis) has been elicited, and if loss of taste is present also, the lesion is above the nerve to the stapedius muscle and distal to the geniculate ganglion. The *nervus stapedius* leaves the facial nerve at the upper end of the knee of the Fallopian canal.

If the lesion is internal to the geniculate ganglion, there will be loss of taste and hyperacusis and loss of tears. The diminution in or loss of tears may be tested by the use of weak ammonia as an inhalant. In the case presented the patient gave negative responses to the tests for taste. He did not complain of hyperacusis. The fracture observed at the decompression operation of the facial nerve passed through the centre of the knee or bend of the Fallopian canal, immediately distal to the *nervus stapedius*.

To finalize the clinical investigation, the function of the seventh nerve and facial muscles is tested by electrical stimuli—faradism and galvanism. The responses are really of relative importance, depending absolutely on the length of time which has elapsed since the accident. Nerve responses to faradic stimuli will be absent in immediate facial paralysis, and absent within fourteen days in delayed paralysis. Muscular responses to galvanic stimuli vary from brisk reactions in some muscles to sluggish reactions

in other muscles. I have found it most difficult to give a definite time for the appearance of the reactions of degeneration in paralysed muscles. The clinical picture, together with the period of time that has elapsed since the accident, remains the better guide.

In all cases of delayed facial paralysis following a fracture of the base of the skull, a decompression operation of the facial nerve is the method of choice. I realize that there exist conflicting opinions as to the most appropriate time for surgical interference.

In my opinion, the decompression operation should be performed towards the end of the sixth week. I gave my reasons in my book on facial paralysis (1950a) and in my article in this journal (1950b), but for emphasis I shall state them again: (i) to permit fibrosis of the blood clot in and around the fracture lines; (ii) to observe any signs of clinical restoration of function in the nerve; (iii) to allow firm healing to seal off the operation field from the possibility of intracranial infection.

If there is any surgical interference within the period designated, the surgeon will encounter free hæmorrhage and unorganized clots which render the decompression operation most difficult—especially the persistent bleeding. The unresolved blood clot around the fractures is a gelatinous mass, bleeding easily at the base and masking the surgical landmarks.

In the case presented delayed facial paralysis was present, and the radiological films demonstrated a gross fracture of the base of the skull involving the mastoid process, squamous temporal, occipital and petrous temporal bones. It was decided to perform a decompression operation when the bleeding from the ear ceased, provided that the patient was considered well enough for the operation. The right ear was completely dry at the end of the third week, the patient was permitted to get up, and at the end of the fourth week the operation was performed.

A post-auricular approach was used, and a transverse fracture of the mastoid temporal bone was encountered at the level of the knee of the Fallopiian canal, passing forward at the junction of the posterior and superior external meatal walls, and through the middle ear to the anterior external meatal wall. The mastoid temporal bone was driven into the skull for 2.5 millimetres. The incus was dislocated into the attic and imbedded in blood clot on the lateral semicircular canal. The middle ear was full of blood clot; some clot was removed with difficulty. The malleus and stapes were uninjured. There was a depressed fracture of the *tegmen tympani*, or roof of the attic, the whole roof being hinged to the outer wall and adherent to the *dura mater*; removal was impossible. The Fallopiian canal was decompressed up to the fracture at the knee of the canal. At this point it was most difficult to decide which was the sheath of the facial nerve and which unorganized fibrous tissue.

The sheath was incised from the stylo-mastoid foramen to the fracture. The bleeding now was serious, and it was decided not to decompress the Fallopiian canal from the attic, so I closed the wound.

At the end of ten weeks no clinical improvement could be detected in the function of the facial muscles, and reactions of degeneration were present in all muscles. I was suspicious of the site where the fracture involved the knee of the Fallopiian canal, and considered that a further exploration was indicated. The patient agreed, the wound was reopened, and I decided to decompress the Fallopiian canal from the geniculate ganglion to the site of the fracture in the knee of the Fallopiian canal in the medial wall of the aditus.

The dislocated incus was removed with old blood clot, and more clot was removed from the middle ear and attic; the depressed roof of the attic was left *in situ*. The facial sheath was incised. The facial nerve above the fracture was discoloured and oedematous, bulging out of the facial sheath like an earthworm. I carefully carried the incision through the fibrous tissue, which was caught up in the fracture at the knee of the Fallopiian canal, and found the nerve completely pinched by the upper and lower fragments. The nerve had been carried into the skull by the

lower fragment, which was driven in 2.5 millimetres. This displacement of the lower fragment is demonstrated by the X-ray films. The nerve was intact, but appeared extremely wax-like when carefully lifted out of the facial sheath in the fracture and inspected under magnification with a dissecting microscope.

The nerve was gently relaid in the facial sheath, which was also freed from the pinch, and the wound was sutured, a drainage tube being inserted in a distal stab wound. The patient was discharged from hospital on the tenth day. At the end of three months, tone was evident in *musculus orbicularis oris*, *musculus risorius*, *musculus zygomaticus* and *musculus quadratus labii superioris*. At the end of six months, on clinical examination muscular function was obvious in all facial muscles, with the exception of the frontal muscle.

Report of a Case.

S.S., a male patient, aged twenty-seven years, a wharf labourer, while unloading a vessel was struck on the side of the head by a packing case, which weighed approximately one ton, in a sling. He does not remember any other facts until he regained consciousness in hospital. (The patient was unconscious on his arrival at hospital.)

Examination revealed hæmorrhage from the right ear, and a contusion behind the right ear over the mastoid temporal region. He complained of severe headaches, tinnitus in the right ear for nearly three weeks and deafness of the right ear. On the second day after the accident he noticed that drinking was difficult, and he experienced trouble with his speech followed by inability to close his right eye. The headaches diminished, but the tinnitus persisted and was constant.

I examined the patient on the seventh day; he had developed a complete massive right facial paralysis. The deafness of the right ear, of conductive type, was pronounced. There was blood in the right external canal. It was found, on careful examination, that the tympanic membrane was discoloured and a tear was observed involving its annular attachment on the posterior and inferior walls. The middle ear was filled with blood, and a blood-stained discharge oozed through the lacerated tympanic membrane. He did not complain of loud noises. His responses to tests for taste on the anterior two-thirds of the tongue and tip were negative. He was most cooperative.

The operations performed on this patient have already been set out in detail.

A plastic hook was devised to support the sagging muscles. Massage and galvanic electrical stimuli were given three times a week during the entire period of treatment. The galvanic stimulation was discontinued when reasonable clinical movement of the facial muscles was observed.

His mental outlook was not good in the early stages; I found it very necessary to give him "pep talks" at about the third month. At the end of this period, when he discovered for himself that there was function in some of the muscles around his mouth, his mental reaction was immediate and displayed itself by an immature smile.

It is now possible to state, after eight months, that he has recovered complete function in all muscles, with the exception of the frontal muscle.

Acknowledgements.

This case is presented from the Department of Otolaryngology, Sydney Hospital.

I wish to thank Dr. Assheton Chin, of the Dental Department, Sydney Hospital, for his cooperation in this case. My thanks are also due to the staffs of the Department of Radiology and the Department of Photography, Sydney Hospital.

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ILLUSTRATIONS TO THE ARTICLE BY DR. J. PARKES FINDLAY.

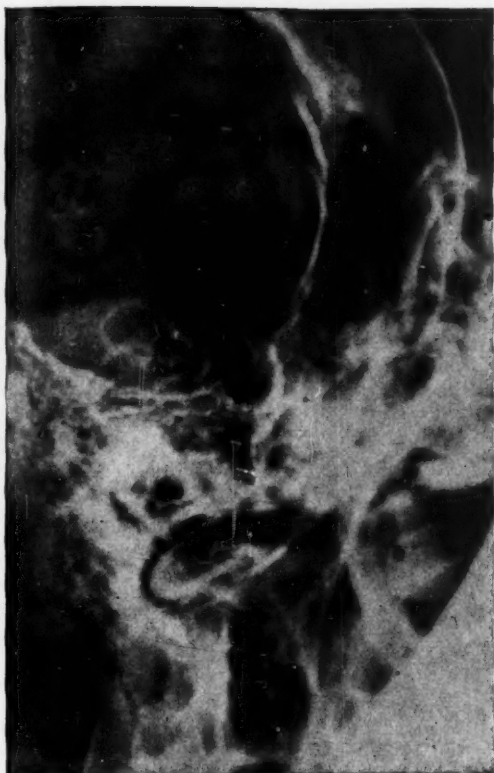


FIGURE I.
A direct lateral view of the skull (right side), showing fracture of the base involving the right occipital, mastoid temporal and squamous temporal bones.

FIGURE II.
A lateral oblique view (right side), showing the fracture as in Figure I, but with the lower fragment of the mastoid temporal bone overlapped by the upper fragment. (This view was taken with the X-ray tube tilted 20° caudad.)

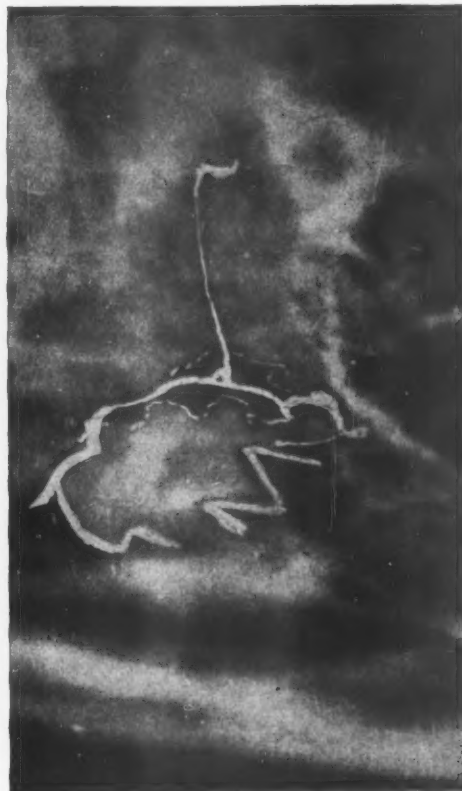


FIGURE IV.
A vertico-submental view, magnified with the fractures marked for emphasis.



FIGURE III.
A vertico-submental view of the skull, showing depressed fracture of the tegmen tympani (right side) of the middle fossa, and a longitudinal fracture extending towards the apex of the petrous temporal bone.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. PARKES FINDLAY.



FIGURE V.

A lateral oblique view magnified to show the lower fragment driven into the skull.

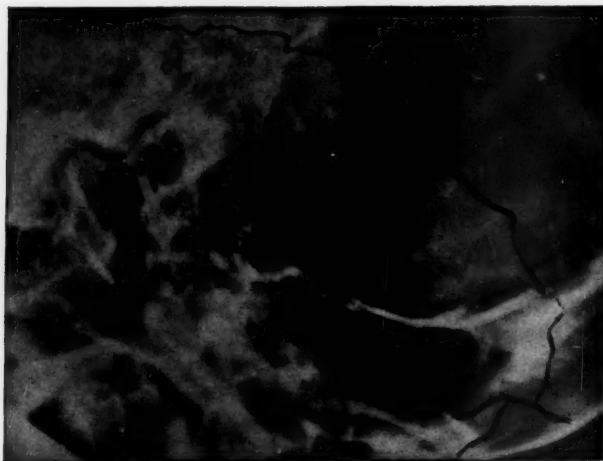


FIGURE VI.

A lateral oblique view magnified with the fracture marked in for emphasis.

TIDAL DRAINAGE: AN EFFICIENT APPARATUS.

By NOEL J. BONNIN, M.S., F.R.C.S.,
F.R.A.C.S.,
Adelaide.

THE frequent automatic irrigation afforded by a tidal drainage apparatus which is working properly, provides by far the best local treatment for "dirty" cystitis and the best method of keeping the bladder clean when prolonged drainage is required.

The apparatus to be described is a modification of that of McKenna. It is self-contained, totally enclosed and compact. The greatest advantage it has, however, is that it works, and works in the general wards in the care of average nursing staff not specially instructed in its use. This is not to say that other types of apparatus do not work, but they seem to require the care of someone with a considerable understanding of hydrodynamics, something of the touch of Merlin, and a deep paternal feeling for the machine. In consequence many of us have given tidal drainage away in despair, and this is a pity. In 1948 I saw this device of McKenna's at a meeting of the American Urological Association, and on my return to Australia I had a copy made. This is illustrated in Figure I. To make it required the services of a skilled glassblower and before long it was broken. It is, I think, too fragile for routine ward use. Moreover, the insides are sealed and hard to keep clean. However, this model did have the advantage, new to me, that it worked and worked very well while it lasted. The model I am now using can be made easily and the materials are usually available in any hospital laboratory. It seems to be sturdy enough for use in the wards, and in the event of accidents, being home made it is soon repaired. It is readily dismantled for cleaning. It is illustrated in Figure II.

How the Apparatus Works.

Figure III shows how the apparatus works. Lotion drips through the dropper tube A into the reservoir tube B, whence it runs through the outlet tube C into the bladder. As the pressure in the bladder rises with increased filling, so the level of fluid in the reservoir tube rises, and air escapes through the air vent needle D. As the level of fluid in the reservoir tube B rises, fluid rises to about the same level in the up limb E of the syphon. Finally it pours over the arch of the syphon into the down limb F and out into the waste bucket. The syphon continues to empty the reservoir tube and with it the bladder until fluid falls to the point at which air enters the syphon tube. This breaks the syphon, and the fluid remaining in the syphon and down tube empties into the bucket, and the filling stage starts again.

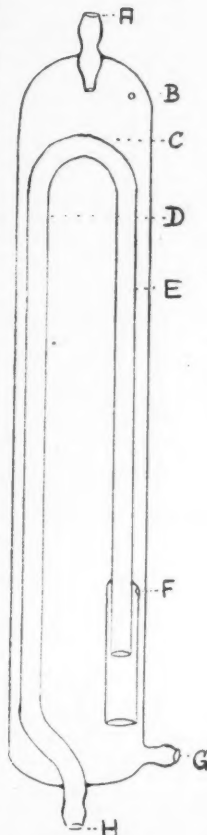


FIGURE I.

Apparatus of McKenna: A, inlet and dropper; B, air vent; C, arch of syphon; D, proximal arm of syphon; E, vent of syphon breaker; G, outlet to bladder; H, outlet to waste.

So far the principles are those of the usual type of tidal drainage apparatus. Now come the differences. Firstly, the reservoir tube B holds a considerable volume of fluid so that it takes quite a time to empty. This gives the bladder a chance to empty adequately, which the usual type of apparatus does not do. Secondly, the syphon breaks cleanly and there is no long period of bubbling and dribbling in the syphon tube. The reason for the clean breaking of the siphon is McKenna's ingenious syphon breaker (G in Figure II). A sleeve of glass over the end of the syphon tube makes a capillary space between the tubes. Capillary attraction holds water here until the water level in the large tube B is well below the syphon breaker vent H. Then, before the water level reaches the bottom of the sleeve tube, the water seal snaps, allowing unimpeded flow of air into the syphon. This syphon breaker makes all the difference between smooth working on the one hand and bubble and trouble on the other.

The duration of the emptying period depends on the rate of inflow of air through the air vent. The smaller this air vent, the longer is the period of suction applied to the bladder. However, the air vent must be large enough to allow air to escape during the period when the tube B is filling. In the model described here a hypodermic needle is used, and size 24 or 25 will be found about right. However, I find that it is an advantage to use a larger needle (21 or 22) and to pack the butt of the needle with cotton wool. This gives the necessary air resistance and seems less liable to block.

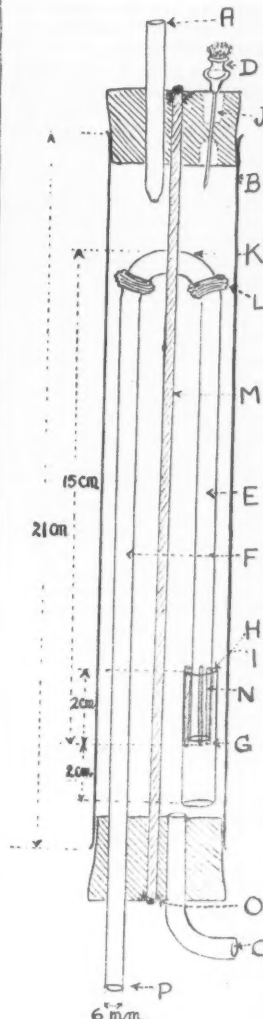


FIGURE II.

A, inlet and dropper; B, reservoir tube (four-centimetre tubing, "Pyrex" glass); C, outlet to bladder; D, air vent needle; E, up limb of syphon (six-millimetre tubing); F, down limb of syphon; G, syphon breaker (sleeve one-centimetre tubing); H, syphon breaker vent; I, air gap, approximately one millimetre; J, arch of syphon; K, rubber cushion (rubber paper band rolled and twisted); L, rubber retaining band to hold stoppers in place; M, rubber strips; N, groove in stopper for retaining band; O, outlet to waste.

Setting up and Operating the Apparatus.

As a rule the apparatus is set so that the bottom of the syphon tube is at the level of the patient's anus, which is the level of the bladder base. In this position, at the end of the filling cycle when the fluid level is at the top of the syphon tube, about 15 centimetres of water pressure will be applied to the bladder. If the bladder is spastic the apparatus may be set higher. It should not be set lower, or the bladder will not be sufficiently filled for an adequate wash.

The waste tube should have an internal diameter about that of the syphon tube. "Soluvac" tubing does very well.

The air vent needle must be quite dry. If it is wet, water will be held in the lumen of the needle by a capillary force more than sufficient to close the needle completely against the pressures employed in the machine. If the apparatus has been sterilized by boiling, then before it is set in operation it is advisable to dry the needle by temporarily attaching to it a dry syringe and drawing air in and out until the lumen of the needle is dry.

The irrigating fluid should be run in at a rate of about 60 to 100 drops per minute; 60 drops per minute will irrigate the bladder somewhere about once an hour, the time depending on the bladder capacity. The rate can

The modification suggested here is easy to make, works equally well and is less liable to breakage. Three thin strips of rubber are required about 10 centimetres long by about two millimetres by two millimetres. Suitable rubber paper bands will serve the purpose. They should be of square section so as to occupy as little as possible of the capillary space. The strips are first passed through the glass sleeve and then one set of three ends of the rubber strips is disposed symmetrically about the syphon tube and held in position there. The other three ends are pulled firmly so as to stretch the rubber right out. The bands become thinned and the glass sleeve can now

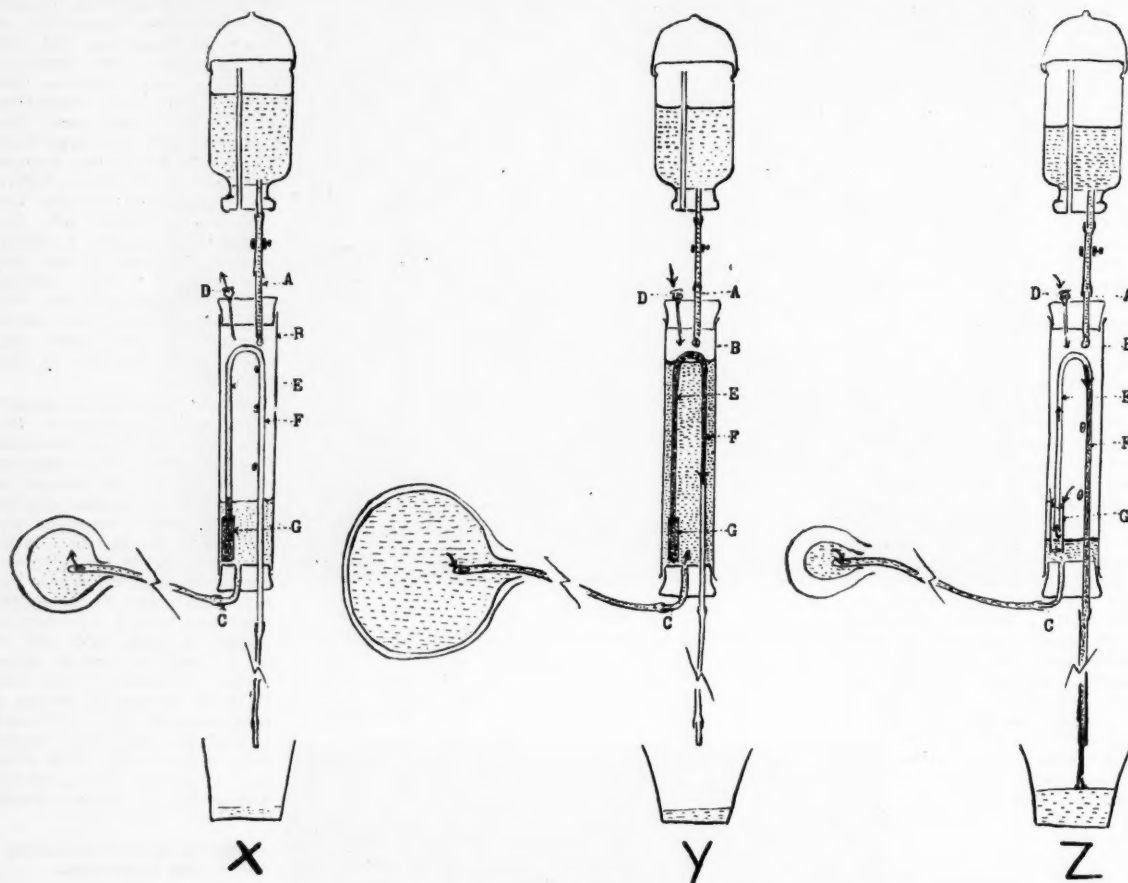


FIGURE III.

X, filling; Y, syphon starts, emptying begins; Z, syphon breaks, emptying ends.

be slower if desired, but if the rate is set too fast, then air may not leave the air vent quickly enough and the syphon will overflow too soon.

Constructional Details.

Specifications shown in Figure II are almost complete and require little elaboration. The sizes of tubing shown should be followed rather closely. "Pyrex" or a similar type of glass should be used throughout. Sodium glass of the size of the large tube is not easy to cut and it does not stand up well to boiling. McKenna's syphon breaker is made by fusing the upper end of the sleeve tube to the syphon tube and then blowing a side hole in the sleeve. This is work for a glassblower and the result is fragile.

be slid into position over the end of the syphon tube. The bands are now released and the rubber shortens and thickens, fixing the sleeve tube firmly in place and at the same time providing a protective cushion between the tubes. The loose ends of the rubber bands are cut away. The rubber stoppers at each end are held in place by a strong rubber paper band as shown. Grooves cut in the stoppers prevent the retaining band from slipping. Rubber cushions, made of coiled-up rubber bands, near the arch of the syphon prevent breakage when the apparatus is being dismantled.

Reference.

McKenna, W. F. (1948), "A Simple, Efficient, Automatic Tidal Drainage Apparatus", *The Urologic and Cutaneous Review*, Volume LII, page 18.

DR. GEORGE OWEN WILLIS, A PIONEER
QUEENSLAND SURGEON.

By E. A. Cook,
Beecroft, New South Wales.

It has been recently pointed out in the journal that as time goes on, the recording of accurate history of the earliest pioneers in medicine in this country is becoming more and more difficult, owing to the absence of written records and the decease of their contemporaries. This fact was forcibly brought to my notice when, some years ago, I set myself the pleasurable, personal task of obtaining the earliest historical records of a small Queensland country town with which I and my father have been closely associated for some fifty years. At first the real importance of such a collection of facts, especially as it deals with the least regarded section of the art of medicine, the unconsidered trifles of every-day practice, seemed so slight as not to be worth the trouble to discover and of no possible value and little general interest. Curiosity, however, induced me to make a few tentative inquiries in the town and a casual search for early documents, and in both I was met with a complete blank. No one remembered anything of the early days and the records had been lost. This put point to the remarks quoted above and stimulated my interest to the extent that I have at last unearthed a tangible history, after seeking information across the world, and unearthing documents nearly a century old in pursuit of a wraith-like figure, which gradually materialized into a vigorous, colourful personality—Dr. George Owen Willis. This, then, is my apology for the following article.

The focal point of this personal account lies in the town of Barcaldine, Queensland, and a brief history of its origin may be of interest. A period of some sixty years elapsed from the formation of the colony of New South Wales by Governor Phillip in 1788 before any white man set foot in this particular district. In the year 1845, Sir Thomas Mitchell, searching for a river running westerly into the ocean, discovered the Barcoo, and a few years later Kennedy confirmed this; but he proved that this river did not run into the Gulf, but lost itself in the centre of the continent. Thirteen years afterwards, in 1858, Augustus Gregory followed the same route up to the Thompson River and then retraced his steps. Another four years elapsed and then Landsborough, making for Brisbane overland, after searching for Burke and Wills, crossed the headwaters of the Thompson, following it to the Barcoo, and on May 21, 1862, arrived at William's station on the Warrego.

Another search party under a superintendent of native police from Rockhampton, Frederick Walter, also set out in search of Burke and Wills on September 7, 1861. On September 27 he reached the Barcoo, which he followed for three days, finding traces of Gregory and Leichhardt. From the Barcoo he crossed to the Alice River, then to Careena Creek, and on northward to the Flinders. This man then probably crossed the actual site of Barcaldine and was the first white man to see and travel through this district. The glowing reports brought back by these expeditions soon resulted in the occupation of the land. At first it was taken up in huge undeveloped areas such as Mount Cornish, Bowen Downs and the like. Later, smaller, more manageable areas were selected such as Corcena and Barcaldine Downs.

These areas gradually became more defined and further subdivided until the early 1870's, when we find two small centres of population appearing—Aramac to the north with Blackall in the south. These towns with Tambo and Clermont comprised the only centres of population at this period in the central west of Queensland and were situated on the original route which, commencing at St. Lawrence, moved inland towards the centre of the continent.

Things remained in a very primitive state with a scanty and scattered population, until the commencement of the central railway extension of the Rockhampton to

Westwood line, which brought a stream of people in its wake to try their fortune in the new country. The railroad reached Lagoon Creek and was opened for traffic in November, 1886, and then the town of Barcaldine was surveyed and building commenced—sixty-three years ago this year, 1949.

At this period the population was almost entirely masculine. It consisted in the main of energetic devil-may-care young fellows, out to seek their fortune. The whole world was theirs for the taking, with almost complete lawlessness and no relaxation, but with hearty and heavy drinking. They were stockmen and bushmen who carried their swags from station to station, peripatetic shearers signing on by contract individually at the big sheds, shed hands and bush workers, carriers to the railhead, drovers, and finally, the constructional workers on the railway line. The little townships which sprang up for a brief existence at each stop of the railhead, such as Pine Hill, Jericho, Alice and others, were composed chiefly of hotels and drinking shops. As many as twenty were licensed at one period in Barcaldine alone. It was a man's world. The wild licence, inseparable from conditions such as this, lasted for some little time, but as the railhead gradually moved on and finally halted in the centre of the western plains, law and order regained control, and the small community set itself seriously to the task of settling down to solid work and home building. In connexion with this, I quote the leading article of *The Champion*—the local newspaper—for January 1, 1892, some six years after the foundation of the town. It says:

That section of the colonists of Queensland who had selected each successive terminus of the Central Railway line for the locality of their business operations, may now be said to have forsaken the shiftless, nomadic system of living which was forced upon them by the necessity of having to shift camp so often, and are now establishing themselves permanently along the line from Alpha Creek to the Thompson River, selecting those sites which after due consideration, appear to offer the best opportunity of doing business. These points on the railway line or depots for the inland country, are seven in number, two of which are simply receiving places for the wool which at certain seasons is delivered there from the Barcaldine and Saltern Creek runs—at Saltern railway station and from Delta and Evora at the Alice railway station—of the three towns on the western downs country we presume Longreach will take the lead. It possesses all the advantages of an incomparable site, close proximity to permanent water, and for many years to come will probably retain its position as the western terminus of the Central Railway.

Thus the editor of *The Champion* surveyed the prospect at the end of 1891. The famous strike of that year had ended in July, the year had seen bountiful rain, the line has been pushed on to the Thompson River, and the magical supplies of artesian water were just beginning to be tapped.

It will now be necessary for us to retrace our steps and deal in more detail with these five or six years during which the town had settled down to sober work and left behind for ever the lawless, careless days of "shifting camp".

The first reference to the proposed cottage hospital occurs on May 10, 1887, six months after the foundation of the town. On this day, Mrs. A. M. Francis, wife of the resident police magistrate, together with a ladies' committee, met at the Court House, and inaugurated a scheme for a series of entertainments to be held during the first week of Queen Victoria's jubilee year.

The tide of settlement flowed into the central district from the north and south simultaneously. The earliest settlers followed Mitchell and Gregory up the Warrego, over the Divide, about the present position of Tambo township, and so on to the head waters of the Barcoo, which was followed down to its junction with the Thompson. From the northern end the pioneers worked westward from the coast along the St. Lawrence, Copperfield Road, and on to Bowen Downs and Mount Cornish centering around Aramac. In the middle 1870's, two hospitals had been established, one at Tambo and the

other at Aramac, the respective centres of population. About 1882 the Blackall hospital came into existence; so that by the time Barcaldine had been founded there were hospitals north and south within a radius of roughly forty miles to the north and seventy miles to the south.

Naturally at a time when all means of travel was by means of the horse over unmade bush tracks, the difficulty and danger of transporting critically ill persons were immense and impossible in wet weather. To quote *The Champion* at this date, November 15, 1887:

The building of the Victoria Cottage Hospital is already being proceeded with, and the contractors hope to have it ready for occupation before Christmas. As its name denotes, the building will be a "cottage" hospital, affording accommodation for 6 male patients. The idea of providing such a hospital was for the benefit of working men, principally those engaged upon the construction of the railway and from the surrounding stations, who had met with accidents or were suffering from severe sickness. Hitherto, such cases have been received into public houses . . . which are not altogether suitable as resting places for invalids. We have now in Barcaldine, a practitioner of high attainments, and it is to the interests of the town and district that his services should be retained. . . . Meanwhile the ladies who have initiated and brought to a successful conclusion the foundation of the institution, deserve the hearty thanks of the community, and we may hope to see them still zealous in working up the funds of the institution, and doing those little offices for the patients which none can do better. . . . In connection with the hospital we must mention Mr. F. R. Murphy's appeal for assistance from the Government, as reported in the Proceedings of the House. Mr. Murphy said he wished to make an appeal to the Colonial Secretary for assistance to the newest hospital in the newest township in the colony, namely Barcaldine.

Two years earlier there was not a single building in the place, but since the extension of the central railway, the place had become a township of considerable importance. As a result of his efforts the sum of £1000 was donated to the hospital, and a subsidy was granted of £2 for each £1 subscribed.

On December 20 the ladies' committee had succeeded in prevailing upon the following gentlemen to act as a building committee to inspect the work of construction. They were Messrs. Francis, Eastlake, Summers and W. H. Campbell. Evidently the ladies had persuaded their husbands to take an interest, despite the contentious fact of having no vote.

Everything was now ready for the opening of the hospital. By February 14, 1888, the building was finished and furnished, the ground fenced in, and all in readiness for the opening on March 1. *The Champion* thus describes the building:

The hospital faces the east and is surrounded on all sides by wide verandahs, the whole set on piles 3 ft. 6 ins. above the ground. It is a remarkably cool place, being open to the fresh north and easterly breezes so prevalent during summer. In the front of the building is the Men's ward which is ceiled, containing four beds. Next door is the Surgeon's room, and on the opposite side the Women's ward containing two beds which will be available for male patients when required. Facing the north is the Matron's and Wardmen's room, having next to it the bathroom into which a tap is placed from a 1200 gallon rain tank which is now full to the brim with good rain water. On the opposite side to the bathroom is the dining room. The kitchen is roomy and detached. The closets are the dry earth system.

Such, then, is the picture presented to the newly appointed Dr. Owen Willis when he arrived from England. At this date he would have been about thirty-six years of age—a tall, dark, strikingly handsome man with a strong personality, a reputation as an excellent surgeon and a renowned horseman, qualities which would make him acceptable in any society, especially that into which he had arrived.

He was born about the year 1852, the eldest son of a Dr. George Willis, who had married a Miss Owen. There were five children of the marriage, of which Owen was

the eldest. The family were born in Monmouth, where Dr. George Willis had settled after leaving his birth place, Fermanagh, Ireland. The family of Willis had a preference for medicine. Dr. George Willis's three brothers were all doctors. Dr. James Willis was a naval surgeon and Dr. William Willis was attached to the British Legation at Tokio. All were tall, dark, heavily bearded, in the early Victorian tradition.

Owen Willis, according to my informant, Miss Rosamund Hutchinson, his niece, was a handsome, heartstrong youth, with something of the wild irrepressible temperament of his Irish ancestry. He was very fond of horses and remained so all his life. His somewhat undisciplined nature was the means of partially destroying a brilliant career, as he became at times very intemperate. References to his drinking habits are constantly turning up—markedly so in the reports of the Barcaldine newspaper, and again in a letter to me from Dr. Vernon W. Padgett, secretary of the Placer Nevada-Sierra County Medical Society, who quoted informants who knew him in Grass Valley, California, where he evidently took to drinking a great deal, and even taking opium derivatives.

He followed in the footsteps of his father and uncles, and went to Edinburgh, graduating in medicine, L.R.C.P., L.R.C.S., in 1876. He proceeded Fellow in 1877 at Glasgow. He was registered in Great Britain on October 12, 1876. He took the L. and L.M. also in 1876 at Edinburgh and Rotunda Hospital, Dublin.

In his graduation year of 1876, he was appointed to the Western Infirmary, Glasgow, for a short time, and later held the post of clinical clerk to out-patients at the Middlesex Hospital, London. These two appointments covering the space of one year constituted his post-graduate resident hospital experience.

In February, 1877, he returned home to his family in Oak House, Monmouth, England, as assistant to his father, and was appointed as surgeon to the Monmouth Hospital which his father had recently helped to found. Later he was appointed district medical officer to the Roachfield District and Monmouth Union.

For six years he practised in the town as assistant and finally partner to his father. He was now about thirty years of age and had apparently been a somewhat boisterous and riotous individual. As an outlet to his naturally impulsive and high-spirited nature, he was wont to gallop furiously about the sleepy village in pure abundance of natural animal spirits. His niece says:

On one occasion his horse bolted in St. John's Street, which is narrow and leads into Minnow Street at right angles opposite an ironmonger shop. Owen and the horse and trap went straight through the shop window and pulled up among the pots and pans.

Emotionally unstable, he quarrelled with his mother because he would go courting Miss Oakley, who later became his wife, in the same carriage and pair which was his father's turnout, used on his medical rounds of the village. There was some difficulty about the proposed marriage. Mr. Oakley, his prospective father-in-law, was a solicitor in Monmouth who lived at Lydac House, some five miles out of Monmouth. The gentleman highly disapproved of the match, evidently noting the grave intemperance and general instability of the young doctor. Mrs. Willis also did not approve the marriage on the belief that they were temperamentally unsuited. The young couple were, therefore, in something of a difficulty, when an old family friend, a Mr. Harris, who was a wealthy bachelor and had an abiding affection for the charming, handsome, headstrong boy, smoothed out the difficulties for the infatuated pair by giving Dr. Owen a marriage settlement of an old-world house known as St. Johns. This house dated from 1620, facing on a narrow cobbled street. The front rooms were small and dark, ceiled with heavy oak beams. The rear had been pulled down in the Georgian period and had tall windows leading out on to a patio and pleasant garden. The consulting room was one of the small dark heavily timbered rooms facing the street. Dr. and Mrs. Owen Willis resided here for about twelve months, during which time he was still practising in partnership with his father.

The family breach occasioned by his unpopular marriage rapidly widened and his relationship with his parents deteriorated, until, at the end of the first year, the couple finally left Monmouth and went to reside at Henley-on-Thames. He left his birth place in 1883, never to return.

The practice at Henley never prospered. Without the backing and restraint afforded by the presence of his father he was unable to carry on successfully. His intemperance grew, and about two years later, in 1885, some three years after his marriage, the final break came. He decided to seek his fortune in the new world, and booked his passage for Australia. His wife refused to accompany him. Old Dr. Willis tried to persuade her to do so by offering to pay her expenses, but she remained adamant, and he was the last to blame her. She returned to her father's house at Lydac where she died in 1939 at the age of eighty-six years.

Dr. Willis arrived in Queensland and was registered as a duly qualified medical practitioner in Brisbane on August 4, 1887. He commenced practice in Barcaldine and was appointed medical officer to the Victoria Hospital on February 29, 1888, some three months after his arrival there. He held this position for fourteen months, resigning on March 5, 1889.

His original appointment was for six months only at a salary of £150 *per annum*. Something of his unstable character must have been noted by members of the hospital committee, for it was decided at the first committee meeting of the new hospital that at the end of six months the position would be reviewed and further applications invited.

Apart from the doctor's drinking habits, it appears that he soon became entangled with the wife of a friend. The local newspaper puts it this way:

He left his wife in England and travelled. In the course of his wandering he drifted to Barcaldine and becoming entangled with another involving the happiness of two friends, he left this part of the world. The lady accompanied him. Her husband remained in Barcaldine for some time, gradually wasting away from the insidious attacks of an internal disease, dying suddenly as he sat on the verandah of the hospital ward.

This unfortunate man seems to have been destined to create havoc and tragedy wherever he went, despite his brilliance as a surgeon and physician.

He was capable of undertaking and successfully accomplishing the most hazardous feats and must have held remarkable faith in his own ability to perform them. To illustrate something of the conditions under which he worked in this new town, I should like again to quote the local newspaper of the period. In reporting the proceedings of the first meeting of the hospital committee, the paper states:

No reply had been received to the advertisement calling for a wardman and Matron, but as the Doctor had an amputation to perform the next day, it was decided to appoint a woman to attend the operation and assist temporarily.

An illuminating decision this. At this time and place a trained nurse was quite unknown. The office of assistant to the doctor at an operation was merely "a woman", any woman who could stomach the sight of plenty of blood and the cries of the patient agonizing under the pain of an amputation done without chloroform. She was at liberty to go when she liked as she was merely appointed "temporarily". This amputation was, however, interesting in that it was the first operation performed in the hospital and almost certainly the first patient admitted. The doctor was out to make as much money as speedily as he could, and undertook the business of a chemist and fancy goods shop as a side-line to the practice of his profession. His advertisements in the local paper enumerate the variety and quality of the goods he had for sale which included "all the best and well-known patent medicines, Toilet and Nursery Requirements, Perfumery, Horse and Cattle Medicines. Dentistry—Teeth carefully stopped, scaled and extracted by a Surgeon-Dentist, Tobacco, Cigars and Cigarettes".

No doubt it was necessary for him to augment his salary of £150 *per annum* as his private practice in a small population would not be excessive. The committee, however, gave him the drug and instrument supply for the hospital in preference to the other local chemist who also tendered, one Oscar Banke.

Thus was launched the Victoria Cottage Hospital, a very modest beginning indeed, but an institution which immediately proved its great value to the small community. The first patient was admitted on March 1, 1888, and by the end of the first month it was obvious that the building was too small for the number of patients seeking admission, and it was decided to enclose portion of the back veranda for further beds and to erect a wash-house and mortuary. These additions were completed on April 24. April closed with five in-patients remaining. On July 17 Mr. J. Lloyd-Jones was appointed secretary to the institution. Later in the same year, September 4, the committee reported that the hospital had become extremely busy and "it is impossible to cope with the number of patients seeking admission. Chronic cases are received for a day or so and discharged to camp on the creek. Men come from as far away as Winton, saying that the fame of Dr. Willis had spread so far and they had heard 'this Victoria Hospital cracked up and the doctor had got his name up, so we came along on our shearers' tickets'".

The Winton hospital, as has previously been pointed out, had been established eight years before in 1880, and remained the only hospital in the far west until that of Longreach was established in 1892. It would seem, then, that the establishment of this hospital at this particular time is due not a little to Dr. Willis's presence in Barcaldine at this period and to his fame as a surgeon. As the contemporary comment in the local paper expressed it, it behoved them as a community to see that Dr. Willis's services were retained. During the six months' term of the doctor's appointment the hospital proved itself an invaluable asset to the district. It was always full to overcrowding with patients suffering from every description of illness to which human flesh is heir. Surgery predominated in the earlier cases and reference is made to operations for aneurysm, removal of eye, splinting of a fractured spine, many amputations *et cetera*, all of which were fully discussed and described in *The Champion* to the greater credit, no doubt, of Dr. Willis, who apparently was as unethical in details such as these concerning his professional prowess as he was lax in his private life and morality. It was not long, however, before there began to appear the dread scourge of these small pioneer settlements, dependent as they were on uncertain and scanty water supplies. Typhoid fever broke out and cases began to come to the hospital. The first reference is to a man, aged sixty-two years, who came in from Saltern Creek and died in hospital from a condition recorded as dysentery. There is no doubt, however, that it was in reality typhoid fever, and the milder term of dysentery may have been used either not to alarm the public or because the diagnosis was incorrect. Certain it is that for many years afterwards Saltern Creek was never without at least one typhoid fever patient, the infection becoming endemic in this area. As summer advanced more cases appeared and accommodation was strained to the utmost. The maximum number of beds was now ten, and as these patients were considered suitable for isolation, it was suggested to equip a tent outside for them, but apparently nothing was done about it.

All was not well, however, with the domestic affairs of the hospital, and it is evident that the doctor believed he might be given his dismissal at the end of the stipulated six months, for in September an advertisement appears in *The Champion* saying that Dr. Willis had let a contract for a private hospital for the convenience of patients from the country, adjoining his private residence. Nevertheless, I can find no trace that this building was ever commenced, and there is no further reference to it.

September passed, and the doctor presumably carried on. In December trouble came to a head. At the monthly meeting on December 11, 1888, the wardman requested

"something extra for fodder for his horse" as he had "to continually run after the doctor on account of the latter not attending regularly which made much extra work for him to do".

Immediately following this complaint the Visiting Committee reported, giving notice of the fact that the surgeon's book was very indifferently written up, and that his attention should be drawn to it. Then the doctor's report was handed in, presumably as the doctor himself was not present. It stated that there were ten patients in the institution and requested that the committee paint the walls. Moreover, "the consumption of liquor had been high but necessary". Here was a pretty collection of debatable points and the committee proceeded to discuss each with some show of feeling. First a paper was presented reporting the doctor's attendance at the hospital which became a matter of lively discussion; in the midst of this another question arose, respecting the appearance of an advertisement in the paper announcing the increasing of the doctor's salary from £150 to £250 per annum, "the document not having been franked by the Committee". The secretary, Mr. J. Lloyd-Jones, pleaded guilty to the action and "had a lively time of it for the next half hour". Mr. Francis considered that the secretary had exceeded his duties altogether in altering anything without consulting the committee, and moreover he considered that it was derogatory to the institution that the Visiting Committee should have so demeaned themselves as to keep note of the doctor's attendance when there was a surgeon's book kept for that purpose. The chairman, Mr. McDermott, then tried to smooth matters over by explaining that "we had practically agreed to raise the doctor's salary to £250". This led to renewed argument and general uproar, and apparently members became so heated that all retired for a time, presumably to take something cooling and allow their ardour to subside.

On resumption there was one thought uppermost in their minds—the question of medical comforts in the shape of port, brandy *et cetera*. The bill for the month was very high—according to the doctor, a necessary increase. The chairman remarked that some better system should be pursued and a check kept. Indeed, there seemed to be a difference of opinion as to whether it was the Victoria Hospital or Victoria Hotel, and another member was "not a bit surprised there were no complaints from the patients if they received all the grog mentioned in the bill for the month". However, after a good all-round discussion, it was agreed that the advertisement should remain announcing the increase of the surgeon's salary to £250. It was then decided to vote £10 10s. "to cover all expenses connected with the testimonial to the ladies who started the institution".

It is obvious from the time of this meeting that the surgeon superintendent was giving no satisfaction to the committee. He did not attend regularly—that is evident—and although Mr. Francis tried to champion him, the members generally were quite decided to remove him at the earliest possible moment. Mr. Francis then proposed Dr. Willis for the position, and moved that his testimonials be read. The motion was negatived. Eventually Dr. Paul was elected, his duties to commence on March 1. In further comment in *The Champion* Mr. Campbell says:

It is only fair to state that the committee has always expressed its approval of Dr. Willis's unremitting attention to his duties, and so satisfied were they with his management that six months ago his salary was raised by £100 per year. Unfortunately, the doctor, who has held important positions in the Old Country, has not yet become acclimatised to the rarefied atmosphere surrounding our colonial aristocracy, gave mortal offence to some of our leading Civil Servants and as a consequence a determined effort was made to deprive him of the hospital with what success our report shows.

The ambiguous references here are, as we now know, to the doctor's drinking propensities and the liaison developing between him and the wife of a member of the committee. Evidently a close friend of the doctor, Mr. Campbell did his level best to save him, despite overwhelming opposition.

On February 12 Mr. McDermott proposed that Dr. Paul's term of appointment be for one year, his duties to commence on March 1, 1889. This motion was carried.

It would seem that this Dr. Paul had already accepted the appointment as the lodge medical officer to the town, and despite the fact that Dr. Willis was still in practice, was duly appointed as surgeon to the hospital. However, the good man must have taken a dim view of the parochial wrangling in this outlandish spot, as we note that he summarily resigned from the position the following month, April 9, 1889. This again left the field to Dr. Willis, who went on his surgical way rejoicing. The local Press was in those days a fruitful field of all and any controversy. Local matters were the main topic and chief interest in a population living practically isolated from the rest of the world. World topics, the destinies of nations and events outside their own immediate neighbourhood evoked little interest, and were scarcely, if ever, reported, but the rivalries of the local medical men were of unsurpassed interest. Not a patient was admitted to hospital, but his every symptom was known and reported in the weekly journals. Accordingly, our publicity-seeking surgeon-apothecary was duly given great space and comment. Not an operation took place but was fully reported for the delectation and instruction of the town generally. For instance: "A skilful operation; a splinter of wood cutting the cornea of a man's eye; the doctor found it would be impossible to save the organ; the patient was submitted to an operation by our own hospital surgeon, who very skilfully extracted the eye; the patient died yesterday." There is a world of speculation here. The fact that the man died, obviously from gross sepsis, was of little moment. The really important thing was the brilliance and daring of the operator who had "extracted the eye". The doctor secured his weekly good Press.

A little later the population was stirred by the report that an operation for laryngotomy had been performed by Dr. Willis and Dr. Hewer, the latter the doctor at Aramack. It appears that the patient had presented himself for relief for a swelling in the throat which was making it difficult to breathe. Says *The Champion*:

The patient on whom the operation for laryngotomy was performed died and it was found that he had an aneurismic tumor of great size in the throat. The operation by Dr. Willis had prolonged life for a short time, but it was impossible to extend beyond a definite period. He had been sub-overseer on Coreena for many years. There being no Minister, Dr. Willis went over to the cemetery and read the burial service.

Times and circumstances, however, were gradually moving to disrupt this congenial partnership, and as has been noted previously, despite the clever backing of the editor and the brilliance of the doctor's surgery, his days were numbered. At this time, April, 1889, Dr. Cumming, who had been working his way north from Tambo, had arrived in the town, and on the resignation of Dr. Paul, who apparently never actually visited the hospital, was appointed official surgeon. Dr. Cumming's credentials were sent by the committee to the Colonial Secretary, as he had not been registered as a medical practitioner in Queensland. He had inserted an advertisement in the paper dated April 2, 1889, as follows: "Dr. W. Cumming, M.B., Ch.B., M.D., Edin., may be consulted at McBrides (hotel) or at Mr. Banks (chemist)", and the following month was actually visiting the hospital "several times a day and night" as the institution was crowded, having fifteen patients, including six enterics. The matron and wardman were assisted by a convalescent patient and a laundress. The monthly report showed that of these fifteen patients admitted, no less than four had died during the month. The good doctor had, therefore, been a sort of freelance and had overlooked the necessity of being registered in Queensland as a medical practitioner, but had been appointed immediately by the distracted hospital committee as soon as he had been seen at McBride's.

The reason is not far to seek, though it may possibly entail a certain amount of supposition. It has been quite apparent that, despite the continual championship of the local Press for Dr. Willis, it would seem that he had not

been attending to his duties. He had evidently commenced with a great flourish of surgery, but had fallen away. It would be unkind to suggest that he had neglected his duties for his overruling weakness, but the inference is plain, and while the hospital surgeon was carousing in the hotel, the hospital was crammed with sick and dying. The presumption is that an epidemic of typhoid fever was raging during this period. Added to the fact of his general delinquency the committee also had to deal this month with an item for Dr. Cumming: £2 2s. for attending an operation and a bill for £17 for medicine supplied, not by Dr. Willis, the hospital dispenser, but by the rival chemist in town, Oscar Banke, at whose shop Dr. Cumming could be consulted. So making virtue of necessity, Dr. Cumming, who had evidently been doing all the work and dispensing at the hospital in the unavoidable absence of Dr. Willis, was asked to take over the official position. The business of chemist, druggist, dentist, oculist, cigar and tobacco merchant and fancy goods emporium conducted by Dr. Willis in direct opposition to the said Oscar Banke had evidently greatly languished, and now Dr. Willis, obviously being hounded from pillar to post, his erstwhile marvelling patients turning against him in his extremity, his undoubted scientific surgery done, had to go. He was formally dismissed from his post at the next committee meeting.

One of the committee men, Mr. Hyland, was moved to speak on the motion of Dr. Willis's dismissal. He said that he had a very high opinion of Dr. Willis as a skilful surgeon, and a good townsman, but he considered the Hospital Committee had a right to dismiss him for the infringement of Rule 30, which prohibited the surgeon being absent for more than twenty-four hours from the hospital without leave of the Visiting Committee. Further discussion on the real reason for dismissing the doctor ensued, and the chairman made the surprising remark that the committee had not dismissed him; he had simply cut the painter and drifted away of his own accord.

On April 15, 1889, Dr. George Owen Willis finally left Barcaldine. He went first to Brisbane for a holiday, accompanied by a lady. He returned alone about two weeks later to take up an appointment as medical officer of the Winton Hospital. As he passed through his old home town on his journey west to Winton he was given a rousing send-off by some thirty-five of his admirers at the Hotel Shakespeare. The following day he resumed his journey by horseback. So he left the town of which he had been the first hospital doctor and prime mover in the foundation of its hospital; he had been in charge of it for a short but tumultuous twelve months. Ahead of him on the Sunday morning heading into the west lay nearly 200 miles of practically uninhabited country with few habitations and immense distances.

What induced this extraordinary man to journey this way? He most undoubtedly loved his horse, and as likely as not it was his sole possession. The railway had not yet been opened to Longreach, but he could have travelled easily and swiftly there by Cobb's coach in a day or so; but we leave him heading into the western sun on his horse still further out—another try to redeem himself and to recover the wasted years, to regain his fast fading ability—another chance to throw off the corrupting influence of his weakness.

Winton Hospital saw him for three short months and he was gone. No further reference is again traceable in any Australian journal.

He now shook the dust of Australia from his feet, and seeking ever new pastures was instantly attracted to the new world of America. He took ship therefore to California, arriving late in 1889, and settled in Alumas County in that State, commencing practice in the village of Grass Valley in 1894.

Here I am indebted to the secretary of Placer Nevada-Pierra County Medical Society of California, Dr. Vernon W. Padgett, for what knowledge remains of this, his final period. Dr. Padgett writes:

I am enclosing two accounts relative to Dr. Owen Willis. One is a letter from a Miss Elizabeth Garland, an old resident of Grass Valley, and another by Sister

Mary Alphonsus of the Convent there, who met the Doctor 52 years ago. Both informants speak very highly of him and his professional ability, but added that towards the end of his life relapsed again into his old habits.

While at first then he was held in high esteem, his weakness pulled him down. Sister Alphonsus writes that he had a small hospital in his residence on Neal Street, and had the reputation of being a very fine surgeon, performing some very difficult operations. He took charge of the Convent Community and Orphanage during a severe epidemic of Diphtheria. "The great Dr. Willis" as he was then called offered his services free to the sorely tried Sisters. In the middle of one stormy winter night, relates Sister Alphonsus, he was called to one of the children suffering from laryngeal diphtheria. The child was cyanosed and nearly pulseless when he finally arrived. Ordering a firm table, a bottle to place under the child's neck, the hired man to hold the child's head, he performed a tracheotomy and saved the child's life. In his death the Medical profession lost a brilliant member.

During his residence in Grass Valley Dr. Willis married a Miss King, who watched and nursed him as he gradually deteriorated until his death on April 12, 1906, at the age of fifty-four.

It is idle to speculate as to what heights of achievement this man could possibly have aspired, had he been blessed with a less voluble intemperate character. That he had ability is undoubted, also a certain courage or perhaps over-daring in his operative work. Wherever he went he made an immediate impression on his patients and the community generally as a brilliant man. The term occurs over and over again. In his sojourn in Queensland and again in California all speak highly of his skill. He must have had a rare presence and a strong personality, with an accompanying fine physique—all the attributes of a successful practitioner. The son of a doctor, with a strong family tradition for medicine, he held everything requisite to make a success of his calling, but failed to achieve anything of note all his life, and instead left a trail of misery and frustration behind him. His is a truly tragic figure, and represents a type of practitioner not uncommon in the "frontier times" at the close of the century. They were the pioneers, however, and as in the case of Dr. Willis, were the founders and originators of the earliest hospitals and medical services in many a western town. They were erratic and unpredictable, both in character and length of stay, but they filled a great want, and brought medical aid and comfort to the sick and ailing, to people completely isolated and without any other means of help. To all these men, now dead and almost all completely forgotten in the fast deepening mists of the past, let us who follow salute them and honour their memory. "*De mortuis nil nisi bonum.*"

CONTROL OF MALNUTRITION.

By I. S. EPSTEIN, M. A. MACKAY¹ and C. N. TURNER,¹

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In a previous paper from this unit, "Some Causes of Malnutrition" (Parsons *et alii*, 1948), the factors responsible for causing a state of malnutrition in a group of hospital patients were presented and discussed. As a result of that study it was thought that further observations on the control of malnutrition would be of value.

With this in view, a group of 33 patients was selected from those attending the Clinical Research Unit at the Royal Melbourne Hospital. Detailed diet histories of these patients had revealed diets apparently deficient in many of the essential nutrients, and the course of each patient had been followed by the unit for a period ranging

¹ Working with the aid of a grant from the Australian Red Cross Society.

from six months to three years, with an average period of twenty-two months.

Of the 33 patients studied, 23 were females and 10 males. In his Birmingham (United States of America) survey, Spies (1947) found the incidence of malnutrition to be higher in females than in males. He attributed this to two factors: firstly, increased requirements during pregnancy and lactation, and secondly, the tendency of the mother in financial stress to restrict her food intake in order that her family might not suffer deprivations. However, these two factors did not explain the preponderance of females over males in this study. Pregnancy did not play a part in any of the cases, and financial stress was shown to be of importance in only two cases. Rather, it appeared that under any stress the female tended to neglect her meals more than the male. She more readily developed faulty dietary habits, which frequently persisted after the disturbing circumstances had disappeared.

The degree to which the diets of the patients were controlled was classified as follows: (i) complete control—when the patient's food habits were corrected and he maintained an optimum diet, as judged by the recommended standards of the National Research Council of America (1948); (ii) partial control—when the patient showed some improvement in food habits, but failed to take an optimum diet; (iii) no control—when the patient failed to improve his dietary habits.

In a previous paper (Parsons *et alii*, 1948), the causes of malnutrition were divided into, firstly, malnutrition due to social factors, and secondly, malnutrition due to organic disease, either as a direct or as an indirect factor. Alcoholism was shown to be the most frequent among the social factors. Other factors were solitary existence, occupation, faulty "therapeutic" dieting, fads and poor food habits. Financial stress was not found to be an important factor in the causation of malnutrition in Melbourne at that time.

It was obvious in the present study that the patient's psychological response to his environment was an important factor in both the cause of malnutrition and the response to treatment. Domestic unhappiness was often associated with one or more factors.

In most instances more than one factor contributed to the cause of malnutrition and difficulty has been experienced in assessing the extent to which each factor operates. However, for the sake of simplicity the cases have been grouped according to the chief causal factor, and associated factors have been noted. These factors and their control will now be discussed.

Alcoholism.

Alcoholism was considered to be the chief cause of malnutrition in 12 cases, six of the subjects being males and six females. In some of these cases other factors had been associated with alcoholism in causing the patients to neglect their diets. Four of them had led a solitary or lonely existence which encouraged neglect of regular meals; four definitely connected their alcoholism with their occupation. Detailed diet histories revealed a typical story of excessive alcohol intake over many years, resulting in loss of appetite, steadily decreasing intake of food and often vomiting of much of the food. All 12 members of the group gave their own story of some alcoholic intake, but in five instances this was far from accurate according to the information obtained from relatives.

Because of the high caloric value of the alcohol consumed, most of these patients did not appear in any way emaciated; frequently their daily caloric intake would be 3000 to 4000 Calories, of which only 500 to 800 Calories were derived from food. It was noticeable that alcoholics showed considerable dislike for sugar and sweets, preferring meats, soups and savoury foods.

Although there was minor evidence of several deficiencies in most of the 12 cases, the principal clinical manifestations of malnutrition were cirrhosis of the liver and peripheral neuritis (four cases), cirrhosis of the liver (three cases), peripheral neuritis (two cases), tremor of hands and tongue (one case), dyspepsia (one case) and severe glossitis and angular stomatitis (one case).

Complete control of dietary habits in this group of 12 alcoholics was effected in only three cases. Each of these subjects had interested relatives taking an active part in their rehabilitation. The patients ceased taking any alcohol and continued to take an optimum diet as instructed. They all made satisfactory clinical progress; one, however, contracted fatal military tuberculosis eight months after coming under observation.

Partial control of dietary habits was effected in four cases. Three patients showed a great improvement in their food intake, but failed to take optimum diets and continued to drink alcohol with apparent control. The improvement in their clinical state was comparable to that of those whose malnutrition was completely controlled. The fourth patient ceased drinking alcohol, but owing to well-established fads failed to take an optimum diet. He had cirrhosis of the liver, splenomegaly and recurrent hæmatemeses. Leno-renal anastomosis was considered to be warranted to lessen the degree of portal hypertension, but technical difficulties prevented the operation proceeding beyond the stage of splenectomy. The hæmatemeses have ceased, but he has recurrent ascites necessitating frequent paracenteses.

No control was achieved in five cases. These patients continued to consume large amounts of alcohol, and appeared to make no attempt to improve their food habits. Two were controlled for short periods when under close supervision by their relatives; when this supervision ceased they relapsed immediately. They have all shown progressive deterioration of their clinical state. In all these cases domestic unhappiness played a part in the failure to achieve control.

Solitary Existence.

Solitary existence was a factor in causing malnutrition in seven cases, five of the patients being females and two males. Because of their solitary lives these patients tended to neglect their regular meals, particularly those requiring much preparation and cooking. They preferred bread, butter, jam, cakes and biscuits because they were more easily prepared and cheaper than a varied diet. Thus refined carbohydrate predominated in their diets at the expense of the protective foods, with a resultant diminution of appetite and progressively smaller meals.

Factors associated with solitary existence in causing malnutrition were alcoholism and faulty food habits; the latter were long-standing and difficult to change. When solitary existence was causing the patients to take an inadequate diet, efforts were made to convince them of the necessity of taking regular, well-balanced meals, although they entailed greater effort in preparation. The patient was advised to seek company for meals, either at home or by dining out.

Of these seven patients, three ceased to live alone and their diets improved considerably as did their clinical state. Three continued to live alone but improved their eating habits (one completely and two partially), while one continued her lonely existence and made no attempt to alter her meal pattern.

With the exclusion of those cases of this group in which alcoholism was the predominating factor, the clinical manifestations in the remaining three were as follows:

CASE I.—M.R., a female patient, aged fifty-six years, suffered from nutritional cirrhosis of the liver with portal hypertension and repeated hæmatemeses. A history was obtained of dietary inadequacy of many years' duration associated with a solitary existence. The malnutrition was controlled only to a minor degree and her condition gradually deteriorated. Twenty-one months after first coming under observation she died of liver failure following a hæmatemesis.

CASE II.—E.M., a female patient, aged sixty years, presented with a hæmatemesis necessitating a blood transfusion. She had never suffered from indigestion and neither barium meal nor gastroscopic examination revealed any evidence of peptic ulceration. Dietary analysis revealed many years of malnutrition. There was no clinical evidence of cirrhosis of the liver and liver function tests gave normal results. The provisional diagnosis was acute peptic ulceration in a patient suffering from malnutrition. With an ulcer régime and correction of the malnutrition the patient's general condition rapidly improved, and she had no further hæmorrhages over a period of six months.

CASE III.—E.H., a female patient, aged forty-seven years, had four attacks of hepatitis over a period of eighteen months. Prior to this her diet had been extremely deficient, particularly in first-class protein and foods of high methionine content. With correction of the dietetic deficiencies, her general condition finally improved, the jaundice faded, the liver decreased in size and the results of liver function tests reverted to normal. She has remained well under observation over the last two years.

Occupation.

Occupation was found to be the chief cause of malnutrition in three cases and associated with other factors in six cases. Amongst the group there were three cooks, a barman, a truck driver obtaining his meals at odd hours on long journeys, a man on night shift whose meal hours did not coincide with those of his family, an office cleaner, and a widow who was too busy to eat more than a "snack" meal as she conducted a shop and cared for a family of three young children. Other factors associated with occupation were alcoholism and poor food habits.

Because of their occupation these patients developed the habit of eating hurried meals at irregular hours, generally at times when no prepared meal was available.

In two instances complete control of the diets was shown when the patients' occupations changed. A third patient continued with his occupation but corrected his diet completely. Six patients had changed their occupation prior to coming under observation, but their faulty meal habits were so well established that they continued to take unsatisfactory diets. However, dietetic education overcame these habits completely in three cases and partially in the other three.

In the three cases in which occupation was the main factor causing chronic malnutrition with loss of weight, two patients had recently developed a peptic ulcer. They both showed satisfactory improvement with a routine ulcer regime and vitamin supplements. The third patient was a cook suffering from malnutrition and macrocytic anemia. Soon after her admission to hospital she developed severe pneumonia, which was treated successfully with penicillin, blood transfusion and liver extract. After her recovery from this illness it was found that she maintained good health when taking a full diet and receiving liver injections. The nature of the anemia is now being investigated.

Faulty "Therapeutic" Dieting.

"Therapeutic" diets may be responsible for considerable malnutrition if they are not carefully supervised and if suitable supplements are not included. The tendency to omit whole groups of foods without substitution can lead to serious depletions.

Failure of the patient to understand his dietetic instructions may also result in his making apparently minor alterations or omissions to the prescribed diet with disastrous effect.

In the present study three patients were found to be suffering from the effects of faulty dieting.

CASE IV.—A.L., a male subject, aged sixty-five years, first attended our clinic complaining of progressive weakness, pallor and loss of weight for twelve months. He had given up work and was leading the life of an invalid. Twelve months earlier he had consulted a physician, who had advised a "meat free" diet for hypertension. The patient kept to this diet so rigidly that he had omitted all meat, eggs and cheese. As a result his protein intake was restricted almost entirely to vegetable protein and his intake of minerals and vitamin B complex was greatly reduced.

On examination at the clinic he was found to have hypertension, arteriosclerosis, albuminuria and hypochromic anemia. The anemia was probably due to chronic hemorrhage from hemorrhoids and low iron intake. With restoration of the deficient factors and oral iron therapy, the patient's anemia was relieved, his general condition improved greatly and he returned to work. Twelve months later he died of uremia consequent on renal damage associated with hypertension and arteriosclerosis.

CASE V.—L.A., a female patient, aged forty-four years, had been a patient in a country hospital for a period of nine months suffering from thyrotoxic cardiac failure with oedema of the legs and varicose ulcers. She had been placed

on a special diet, but loss of appetite and dislike of the type of food served to her resulted in her eating very little of the hospital food. She persuaded her relatives to bring her sandwiches and cakes, which she ate when she felt hungry. Lack of dietetic supervision allowed this state to continue for nine months, when she was transferred to our unit. The patient's diet was then discussed with her, her dislikes were considered and suitable alterations were made. She was found to be most cooperative in taking a strict low-sodium diet. Her cardiac reserve increased, the oedema cleared, the varicose ulcers healed and her mental condition improved. Thyroidectomy was performed successfully and she was discharged from hospital back to the country, ambulatory and well. She has remained well for twelve months, except that she has become excessively obese from overeating and there has been recurrence of the varicose ulcers.

CASE VI.—E.McD., a female patient, aged sixty-seven years, had been given dietary instructions by her physician for hypertension, but had modified these to suit her fancy. Fear of increasing weight and flatulence after fatty foods associated with cholelithiasis caused a further restriction of her diet, so that the resultant intake was most unsatisfactory.

She was admitted to hospital with cholecystitis and cholelithiasis. She had lost one stone in weight in twelve months, but there were no other clinical manifestations of malnutrition. The patient was given a protein-rich diet of high caloric value, her general condition improved, and three weeks later cholecystectomy was successfully performed. In this case faulty "therapeutic" dieting was associated with organic disease which dominated the clinical picture. Correction of the dietetic deficiency in association with therapy directed towards the prime organic disease was an important factor in the patient's satisfactory progress.

Poor Food Habits and Fads.

Eight patients in this series were ill-nourished primarily as a result of their poor selection of foods and long-standing fads. In six other cases poor food habits were a secondary factor, the primary factor being alcoholism, occupation, solitary existence or organic disease.

These patients, particularly the housewives, had developed over a period of years a disinterested attitude towards food. Although they prepared good meals for their families they ate very little themselves apart from bread, butter, jam, biscuits and tea.

The eight patients of whose malnutrition poor food habits were the chief cause all presented with dyspepsia, which itself was partly the cause of the restricted food intake in several cases. The relationship between malnutrition and the symptom of dyspepsia in this group is obscure. In three cases there was associated gastritis, but in the remainder no organic lesion of the gastrointestinal tract could be demonstrated. However, controlled dietetic therapy resulted in complete amelioration of symptoms and satisfactory improvement in every case.

In one case there was definite evidence of avitaminosis as shown by angular stomatitis and glossitis, but there were no other clinical signs of malnutrition.

Eight of the 14 patients in this group continued to take optimum diets, dietary education having been responsible for the change in their food habits. The remaining six patients showed a great improvement in their diets, but were unable to overcome many of their well-developed fads.

Organic Disease.

Organic disease was the direct cause of severe malnutrition in six cases. Less severe grades were commonly encountered, and dietetic correction gave excellent results in most cases. The diseases responsible for grossly deficient diets were gastric carcinoma (two cases), chronic gastric ulcer, cardiospasm, cerebral tumour with vomiting, and duodenal diverticulum.

In these cases energetic correction of the associated malnutrition was an important preliminary step in the improvement of the patient's general condition prior to direct treatment of the organic disease. The measures adopted to achieve this correction included the use of diets rich in protein and vitamins, with the valuable addition of supplementary "chaser" drinks of high caloric value, especially at night (Parsons *et alii*, 1948), and occasion-

TABLE I.
Showing the Extent of the Control of Malnutrition in 33 Cases Studied over an Average Period of Twenty-two Months.

Factor Causing Malnutrition.	Total Number of Cases.	Male Patients.	Female Patients.	Control of Malnutrition.								
				Complete.			Partial.			Nil.		
				Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
Alcoholism ..	12	6	6	2	1	3	3	1	4	1	4	5
Solitary existence ..	7	2	5	—	2	2	2	2	4	—	1	1
Occupation ..	9	6	3	4	2	6	2	1	3	—	—	—
Faulty "therapeutic" dieting ..	3	1	2	1	1	2	—	1	1	—	—	—
Poor food habits and fads ..	14	4	10	3	5	8	1	5	6	—	—	—
Organic disease ..	6	1	5	—	—	—	—	—	—	—	—	—

Dependent on nature and course of organic disease.

ally the intragastric drip administration of milk mixture of high caloric and high protein value (Garlick, 1949).

This realization of acute malnutrition as a concomitant of lesions affecting the gastro-intestinal tract is of considerable importance, and its full correction is an integral part of therapy, particularly as a pre-operative measure.

Discussion.

The clinical manifestations of malnutrition as observed in this group of patients varied from advanced cirrhosis of the liver or peripheral neuritis, or both, to simple underweight and lack of energy without any other specific findings. It should be appreciated, however, that malnutrition may not be accompanied by loss of weight—this applies particularly to chronic alcoholics.

The progress of the clinical state varied with the degree of control of the malnutrition. In the group showing complete control there was considerable clinical improvement in every case. In several cases in which developed cirrhosis of the liver was present, as shown by liver function tests and liver biopsy, there was dramatic improvement in liver function with disappearance of the jaundice, resorption of the ascites and improvement in the results of chemical tests. This improvement has been described in a previous paper from the unit (Wood *et alii*, 1949).

In the group with no control of malnutrition there was uniform deterioration of the clinical state.

The measures taken to eliminate dietetic deficiency were as follows: (a) explanation to the patient that malnutrition was the cause of the illness and that continuation of the previous dietetic regime would result in progressive deterioration; (b) removal as far as possible of causal factors—for example, unsuitable occupation, alcoholism; (c) dietetic instruction; (d) supervision of the patient's progress after discharge from hospital by ensuring regular attendance at the out-patient clinic and by visits to the home.

The results of our treatment in this series are shown in Table I.

It will be noted that the total number of cases exceeds by 18 the number of patients studied, as when several factors were operating in one case they have been recorded separately. Malnutrition was completely controlled in 13 cases and partially in nine, while no control was effected in five. In addition, there were six cases in which organic disease was the direct cause of the malnutrition and in which the control of malnutrition was dependent on the course of the organic disease. For ease of comparison, the graph (Figure 1) is presented to show the percentage control in four of the most important groups.

Summary.

1. Thirty-three cases of malnutrition as determined by detailed dietetic analysis have been reviewed with special reference to the degree of control of the malnutrition which has been achieved.

2. The various causal factors of malnutrition have been analysed separately. Alcoholism was the commonest principal cause of malnutrition and was the most difficult to control. Poor food habits and fads also contributed to the

malnutrition in a large proportion of the cases, but were more easily corrected by dietetic instruction. Solitary existence, unsuitable occupation and faulty dieting were involved in a small number of cases, but they are of considerable importance as they may be overlooked.

3. Of the total number of patients studied over a period ranging from six months to three years (averaging twenty-two months), the malnutrition of approximately

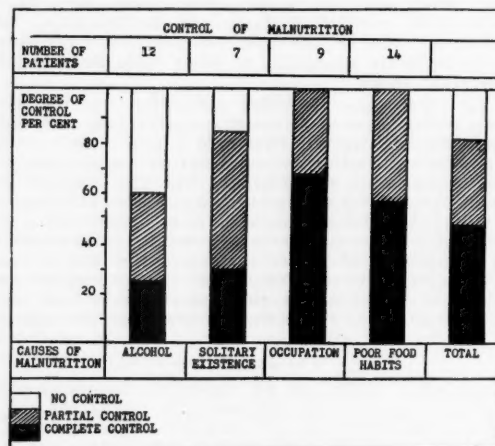


FIGURE 1.

Control of malnutrition. Graph showing the percentage control in the four major causes of malnutrition.

one-third could be controlled completely, that of rather more than one-third could be partially controlled, and that of rather less than one-third failed to be controlled.

4. The clinical progress of the patients tended to vary directly with the degree of control of the malnutrition, but was modified by associated conditions.

Acknowledgements.

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THE SALIVA: A SHORT REVIEW, WITH SPECIAL REFERENCE TO DENTAL CARIES.

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IN the course of studies on dental caries it was found that no satisfactory and easily accessible review of the present-day knowledge of saliva existed; hence, before any experimental work was undertaken on saliva, it was necessary to survey a large and scattered literature. The present summary may help to awaken interest in this important subject and in some measure to make good the apparent lack of a comprehensive review.

Although saliva is one of the largest secretions of the body (1000 to 1500 millilitres daily), surprisingly little interest has been taken in the salivary glands and their secretions. This is borne out by the fact that very little information about these glands, or their secretions, is to be found in most text-books of physiology; still less in text-books of bacteriology, although whole saliva contains many varieties of microorganisms and itself appears capable of supporting the growth of certain microorganisms (Goldsworthy and Florey, 1930).

In recent years, however, investigators in the field of dentistry have emphasized the relevance of saliva to the urgent problem of dental caries. Saliva is clearly an important constituent of the gross environment of the teeth, and, as more and more knowledge is gained of the properties of tooth structures and the process of dental caries, saliva is assuming a new significance in the study of oral health and disease (Karshan, 1938).

ANATOMY AND CYTOLOGY OF THE SALIVARY GLANDS.

The salivary glands consist essentially of three paired masses of cells, the parotid, the submaxillary and the sublingual. There are also a number of small glands scattered over the mucous surfaces within the buccal cavity; they secrete mucus only.

The individual salivary gland comprises a number of alveoli, each having a single layer of cells enclosing a central space or atrium. The secretion is emptied into these atria, which drain into small ducts. Several of these ducts combine to form a common channel, which empties into the large excretory duct, itself opening into the mouth. The parotid duct opens on the buccal surface of the cheek opposite the second upper molar. The openings of the submaxillary duct and of the several smaller ducts from the sublingual gland are situated near the frenum of the tongue.

The cells forming the alveoli of the parotid gland are entirely serous in nature and secrete a watery fluid containing organic substances, including protein bodies and inorganic crystalloids. The submaxillary and sublingual cells are of two types: some are serous, as in the parotid, and others are mucous cells, secreting a viscous solution of mucin in water. The small buccal glands are all mucous.

The cells of the glands appear granular in structure owing to the presence of minute colloidal droplets, which increase in number when the glands are resting. In the serous cells these granules are considered to contain the enzymes secreted in the saliva and are termed "zymogen" granules. The "mucinogen" granules in the mucous cells are coarser and are believed to be broken down to mucin when the cells receive certain stimuli.

PHYSIOLOGY OF THE SALIVARY GLANDS.

The increase in oxygen uptake of the cells in the salivary glands during their activity indicates that their function is not merely one of passive filtration but one of active secretion. Secretion of the glands is activated by nervous stimulation. (The immediate activator is chemical and thought to be acetyl choline.) In animals some hormonal stimulation may occur, but this is very rare in man. The

nervous stimulation is effected by two reflexes, either the "conditioned" reflex, when some sense organ other than taste is stimulated, or the "unconditioned" reflex, when the nerves of the mouth associated with the sense of taste are stimulated by the presence of food. These secretion-reflexes may be inhibited by muscular activity, mental work or emotional strain, such as fear.

With the "unconditioned" reflex the type of stimulation depends on the nature of the food which stimulates the taste organs. The salivary glands appear to respond selectively to different stimuli. It is thought that various agents coming into contact with the mucous membrane of the mouth cause (through the salivary centre) stimulation of specific groups of epithelium. These cells apparently possess either their own specific secretion-mechanisms or else a differential permeability to the constituents of the blood, and are able to alter this permeability according to the nature of the stimulus. The degree of alteration depends then on the strength and type of stimulation, and this results in the secretion of saliva of the type required by the body for each particular purpose or occasion. For instance, cold water, ice or physiological saline scarcely gives rise to any effective stimuli, yet an entirely inedible substance, such as a pebble or sand, can cause very active salivation. In the first example the body does not react either favourably or unfavourably to the water, ice or saline. However, in the second example pebbles or sand are not digestible, but the glands secrete large quantities of thin saliva to facilitate their removal from the mouth. Again, when caustic substances are taken into the mouth, the salivary glands secrete a thick mucoid saliva, thereby giving protection to the oral mucosa.

FUNCTION OF SALIVA.

The salivary glands can, as discussed above, adapt their secretion to a variety of functions. Saliva can assist in digestion, not only by supplying the enzyme ptyalin, but also by lubricating food for swallowing. It can, in times of disease, act as an excretory mechanism for urea, sugar and poisons, and has been reported to be able to assist in maintaining the water-balance of the body. Saliva also carries out the important functions of moistening and lubricating the soft parts of the mouth and lips, cleansing the teeth and inhibiting the growth of certain pathogenic and saprophytic bacteria.

COMPOSITION OF SALIVA.

Chemical Constituents.

Saliva has no fixed composition, because the salivary glands have the ability to alter the permeability of their secretory cells and the quality of the secretion. Qualitatively saliva resembles blood plasma in composition, many of the same organic and inorganic compounds being present in both fluids. Analysis of saliva expectorated from the mouth—that is, saliva including bacterial flora—reveals that water forms 99.5% of the total weight of saliva; the remaining 0.5% of solid (Babkin, 1938) consists of ions of sodium, potassium, calcium, magnesium, chlorine, carbonate, phosphate and thiocyanate (Rapp, 1946); and the organic compounds of mucin, serum-albumin and globulin, amino acids (Kirch *et alii*, 1947), urea (Stephan, 1940), uric acid ("Notes, Comments and Abstracts", 1939), citrate (Shulman and Robinson, 1948), creatine and phospholipides (Krasnow, 1945). The gases oxygen and carbon dioxide are also in solution. Saliva contains a number of enzymes; whether these are all secreted from the salivary glands is doubtful, because there is little evidence that analysis of saliva has been carried out on sterile saliva direct from the gland-duct. Most analyses are carried out on the mixed or whole saliva of the oral cavity, which supports a heavy bacterial population rich in enzymes and the products of bacterial metabolism. Earlier analyses have revealed the presence in saliva of an amylase (ptyalin), maltase, lipase (Eggers, 1946), catalase (Leonard and Kokatmur, 1939), peroxidase, mucinase (Simmons, 1941), urease (Stephan, 1940), lyso-

¹Portion of this work was carried out with the aid of a grant from the National Health and Medical Research Council.

²The term "saliva" refers, in this paper, to the mixed saliva of the oral cavity containing bacteria and cellular debris. If sterile saliva straight from the glands is referred to, this is stated.

zyme (Fleming and Allison, 1922), phosphatase (Broomall, 1939; Glock *et al*, 1938) and reductase (Wessinger and Fosdick, 1937). Rapp (1946) reported the presence of carbonic anhydrase in saliva; however, Anderson (1949) has been unable to confirm Rapp's results. Recent work of Dentay and Rae (1949) shows phosphatase not to be a salivary enzyme but to be present in saliva by virtue of its content of bacteria and cellular debris. Mucunase and ptyalin are the only other enzymes that have been found in duct-saliva. The catalase content of normal saliva was found by Deakins (1938) to be reduced 70% when passed through a Berkefeld filter. The remaining 30% of the catalase may be truly salivary enzyme or may be a bacterial exo-enzyme. The lysozyme in saliva is thought to be produced not from the salivary glands but from the polymorphonuclear leucocytes, which become mixed with the saliva when it reaches the oral cavity. It seems quite probable that reductase, maltase, urease, peroxidase *et cetera*, enzymes which have often been isolated from bacteria, may yet be found to be bacterial in origin and not secreted by the salivary glands.

Bacterial Content.

The bacterial content of saliva of the oral cavity is very large and very varied. Nearly all known bacteria have been isolated from the mouth at one time or another (Bibby, 1939). How many of these are merely transitory has been somewhat difficult to establish. Cocci form more than 50% of the total oral bacteria; these cocci are both Gram-positive and Gram-negative; many are streptococci, mainly of the viridans group; staphylococci and Sarcina have also been found.

Bacilli constitute about 10% of the total oral bacteria; the genus that has received the most attention is *Lactobacillus*, which represents about 2% of the total viable organisms of the saliva. Diphtheroids and spore-bearers seem only transitory; Gram-negative rods are also present in saliva, but few investigations of these organisms have been made, so that knowledge of them is not extensive.

Vibrios, fusiform rods, *Leptotrichia*, *Actinomyces* and fungi have all been found in the mouth in varying numbers. The fusiform rods are the most important of this group, sometimes making up as many as 10% of the total bacterial population.

The numbers of organisms vary continually as between individuals (Bibby, 1938b) and in the same individual from hour to hour and day to day (Hine and Bibby, 1939). Also the method of stimulation of flow of saliva greatly affects the bacterial content of the saliva, because the flora of the mouth varies from area to area in the same mouth; hence the bacterial content of the saliva will be in part or largely dependent on the area from which the bacteria were dislodged.

The Antibacterial Properties of Saliva.

The bacterial content of the saliva is greatly influenced by the fact that the mouth does not support the growth of all bacteria. Moreover, the saliva of itself displays several antibacterial properties. One of the main functions of the saliva is the continual washing of the mouth. Any organism that enters the mouth must have the ability, therefore, either to multiply quickly or to colonize on the mucosa or tooth-surface, if it is to remain in the oral cavity and the saliva. Also, the established flora of the mouth may inhibit the growth of another organism either by a direct antibiotic effect of an inhibiting end-product or by competition for the same substrate (Hill, 1939).

Bibby, Hine and Clough (1938) found that growth of 110 of 169 strains of bacteria was inhibited by saliva. The strains most affected were the air and water cocci and bacilli, and the strains least affected were those isolated from the mouth.

Belding and Belding (1948) claim that a mucoid streptococcus is the organism which is responsible for this antibacterial action of saliva. Bibby (1938a) and Hill (1941), however, claim that a lysozyme, an enzyme which causes lysis of bacterial cells, is the responsible agent. This substance is effective against *Micrococcus lysodeckticus*, *Bacillus megatherium* and *Sarcina*. It will not pass through a Berkefeld filter, is only slightly weakened by

heating at 75° C. for five minutes, but is destroyed by ultra-violet light and storage at 20° C. Another inhibitory compound, which is effective against staphylococci, streptococci, lactobacilli and colon-typhoid organisms, and has many properties similar to those of amylase, has also been isolated from saliva. It passes through a Berkefeld filter, is destroyed by temperature of 75° C. for five minutes, but is resistant to ultra-violet light and storage at 20° C. (Bibby, 1938a).

Hammond and Weinman (1942) claim that phagocytosis can take place in saliva as there are present both viable phagocytic cells and opsonin.

SALIVA AND DENTAL CARIES.

In 1890 Miller (1890) put forward the chemo-parasitic theory of dental caries; this has formed the basis of the most widely accepted explanation of the disease-process. In those parts of the mouth isolated from the mechanical cleansing effect of food and saliva, such as between the teeth or in the pits and fissures of the tooth-surface, mucin (a glycoprotein present in saliva) is deposited on the smooth surfaces of the tooth, enmeshing bacteria, food-debris and epithelial cells in a thin gelatinous or rubbery, transparent film. This structure is the so-called mucinous plaque. Organisms such as lactobacilli and streptococci, which can produce acid metabolites from carbohydrates, have been isolated from these plaques. It is thought that some of the acid end-products diffuse out through the plaque and are washed away, but some remain in the depths of the plaque and attack the calcium phosphate of the enamel beneath. Dental caries results.

Gottlieb (1947) and Pincus (1948), however, believe that proteolytic organisms are also active on the tooth-surface and that the initiation of caries is due to the breakdown of the protein constituents of the enamel. Some workers believe that protein breakdown is accompanied by solution of the inorganic portion of enamel, others that caries is due solely to the breakdown of the organic portion of the tooth. As yet no histological evidence has been presented to show which portion of enamel disintegrates first at the initiation of caries, the protein or the calcium phosphate.

Because acid-production has an important place in these theories, any mechanism which is likely to minimize the effects of these locally produced acids or affect the solubility of enamel in these acids must be of importance in the process of caries. It is known that saliva (as secreted by the glands) possesses acid-neutralizing properties, and in view of the fact that saliva continually bathes the teeth it was felt that a special relationship must exist between dental health on the one hand and the balance between the saliva's ability to produce and to neutralize acid on the other hand. Consequently it was thought reasonable to attempt to determine differences between the saliva of an individual immune to dental caries and the saliva of an individual highly susceptible to the disease. To this end the concentrations of various inorganic and organic compounds, which had been detected in saliva, and the enzyme and bacterial content of saliva have all been investigated.

Investigation of Chemical Composition as Related to Dental Caries.

Inorganic Constituents.

One of the first properties of saliva investigated was the hydrogen ion concentration (Bunzell, 1923; Starr, 1922). As the accepted theory of caries suggested that acid produced in the mouth caused caries, early workers seem to have expected to find that a caries-immune individual had a higher salivary pH value than a caries-susceptible person in whose saliva, also, a lower titratable acidity would be expected, because less acid was presumably present (Hubbell, 1933). Owing to the various techniques used by the different workers, the results of these investigations are somewhat conflicting.

As early as 1915, Marshall (1915) suggested that the neutralizing power of saliva had a strong influence on caries, the teeth of the caries-immune receiving more protection from the saliva. In recent years this acid-neutralizing property of saliva has been more fully investigated and is claimed by Spies and his co-workers (Dreizen

et alii, 1936) to show a good correlation with caries activity.

In 1932 Enright, Friesell and Trescher (1932) showed that the solubility of enamel was dependent not only on the hydrogen ion concentration but on the ionic concentration of calcium and phosphate in the solution. Investigators endeavoured to demonstrate that susceptibility to dental caries was dependent on the calcium and phosphate concentration of the saliva (Karshan, 1938), the person with a higher calcium and phosphate concentration showing greater resistance to caries. Results were very conflicting owing to differences in analytical technique and method of salivary collection. Saliva has no fixed composition, and great variations in concentration of the constituents are found. In the majority of cases the variations in the one patient were as great as the variation between the two groups (immune and susceptible).

Karshan (1935) incubated saliva, sterilized by the addition of mercuric chloride, with solid tricalcium phosphate and estimated the decrease which occurred in calcium concentration in the saliva. A greater decrease occurred in the saliva of caries-immune patients, which Karshan claimed was due to the supersaturated state of the saliva with regard to calcium and phosphate ions. Rae and Clegg (1948), however, have more recently shown that the precipitation of the calcium and phosphate in the saliva-tricalcium phosphate solution was due not to the calcium and phosphate ionic concentrations but to the hydrogen ion concentration of the solution, the solution of higher pH precipitating more calcium.

Organic Constituents.

When satisfactory correlation could not be established between salivary calcium concentration and caries, Krasnow and Oblatt (1935) attempted to fractionate the calcium present in saliva into colloidal and non-colloidal portions. The more stable colloidal calcium, such as in a calcium-proteinate of mucin, was found to be in higher concentration in saliva of caries-immune persons. These workers also investigated the protein content of saliva and its relationship to caries (Krasnow and Oblatt, 1933). They found a higher protein value more often in caries-immune patients and also a more stable type of protein. Karshan (1935), however, found no significant difference between the protein content of the saliva of the two groups of individuals.

Other organic compounds that have been investigated are amino acids, insulin, glycogen and opsonin. Kesel (Kirch *et alii*, 1947) found that the amino-acid content of saliva varied greatly from one individual to another, and there was apparently no correlation with the caries-activity of the individuals.

Turner and Crane (1944), however, claim that lack of tryptophane in the saliva showed correlation with susceptibility to caries; that tryptophane increases the reductive activity of saliva, but partially inhibits the amylolytic activity. Thus in the caries-free, where tryptophane is present, the saliva reduces more quickly but displays less amylolytic activity. Eisenbrandt (1943), in very carefully planned experiments, showed, however, that the oxidation-reduction of saliva was not a function of the individual.

Gore (1941) investigated insulin and glycogen content of saliva, but was unable to establish any relationship with caries activity.

Opsonin content of saliva was investigated by Hammond and Weinmann (1942), who found that an opsonin-inhibiting factor was present in higher concentration in susceptible patients than in immunes. This allowed more phagocytic activity in the saliva of the immune individual.

The principal salivary enzyme that has been investigated is ptyalin. Again, however, results are conflicting. Some investigators (Turner and Crane, 1944) claim, others deny (Hess and Smith, 1948) a significant difference in concentration as between saliva of the caries-free and the caries-active.

Investigation of Metabolism of Oral Bacteria as Related to Dental Caries.

Workers have investigated the bacterial content of the saliva as well as the inorganic, organic and enzyme com-

ponents of saliva in their search for the cause of dental caries. Lactobacilli, aciduric streptococci, diphtheroids, yeasts, staphylococci and certain *Sarcina* are all capable of producing sufficient acid to dissolve enamel, but only lactobacilli show an increase in number in the saliva, when caries activity increases.

Kesel *et alii* (1946) claimed that *Bacillus aerogenes*, an ammonia-producing organism, occurred in immunes' saliva. Earlier workers had endeavoured to correlate the ammonia content of saliva with caries (Grove and Grove, 1934). In 1946 Cary (1946) showed that the ammonia was not secreted from the salivary glands but was produced from bacterial action, and that more ammonia was formed when saliva of caries-immune people was incubated than when that of caries-active persons was incubated.

The acid-producing power of saliva has also been investigated in an endeavour to find a correlation between salivary properties and caries activity. When saliva of a susceptible individual is incubated in glucose agar, acid is produced. Also, if saliva from a caries-active person is incubated with glucose and enamel, acid is produced and portion of the enamel dissolves. Laboratory tests for caries-activity which are based on these acid-producing properties of saliva have given excellent correlation with caries activity (Fosdick *et alii*, 1937; Hadley, 1933; Snyder, 1940), because all the constituents of saliva are taken into account. The earlier tests which were used to measure caries-susceptibility of any individual took into account only one component, such as calcium or hydrogen ion concentration. It is now accepted that caries involves such complex mechanisms that more than one factor is implicated in the initiation, and, if good correlation is to be obtained between results of tests on saliva and caries activity, the complex conditions of the mouth must be imitated as closely as possible.

CONCLUSION.

All the investigations of saliva have greatly added to our knowledge of the caries process. They have also had practical use in preventive dentistry. A count of the lactobacilli in the saliva (Hadley, 1933) gives a sufficiently accurate index of caries activity to make possible the use of the test to study the effect of certain caries-preventive procedures. Jay (1947) has recommended diets with low carbohydrate content as the best method of controlling caries. People adhering to these diets have had regular lactobacillus counts made on their saliva, and the decrease in lactobacilli revealed by these counts has supported the clinical evidence of decreased caries-activity.

The use of many different chemicals in toothpastes, mouthwashes and chewing gum has been advocated in caries-control procedures. These have first been tested by studying their effect on retarding solution of enamel in saliva-glucose-enamel mixtures (Fosdick and Calandra, 1947; Dreizen *et alii*, 1947). If they successfully inhibit acid production *in vitro*, they are then tested *in vivo*. In this way compounds likely to be ineffective are quickly detected. A completely effective compound has not yet been found, but the fluoride ion, vitamin K, indole, iodoacetic acid and ammonium phosphate have all proved moderately effective. Work is still continuing along this line of investigation, and a satisfactory preventive may yet be found.

Further investigations are also being carried out on the ammonia-producing organism, *Bacillus aerogenes*, and on the salivary protein, mucin, and the mucinases. Evidence is being sought to explain the natural immunity of some individuals to the disease and the exact process involved in the initiation of dental caries. Then satisfactory preventives may be developed and this disease, the commonest of civilization, may be brought under control.

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Reviews.

BACTERIOLOGY.

THERE has been a ten years' interval between the appearance of the sixth edition of Professor J. W. Bigger's "Handbook of Bacteriology" and the previous edition. The latter (published in 1939) had of necessity to serve during the war years, but three reprints in rapid succession in 1945, 1946 and 1948 indicate the demand for the book, and, as Professor Bigger himself notes in the preface to the sixth edition, the need for revision and for inclusion of descriptions of the many and rapid advances made in microbiology since 1939. In spite of much additional information, only 80 extra pages have been added, and the book still justifies its reputation for compactness, together with clearly set out subject matter and good illustrations.

In the categorical description of organisms and established technical procedures which any such text-book on bac-

¹ "Handbook of Bacteriology: For Students and Practitioners of Medicine", by Joseph W. Bigger, M.D., Sc.D. (Dublin), F.R.C.P. (London), F.R.C.P.I., D.P.H., M.R.I.A.; Sixth Edition: 1949. London: Baillière, Tindall and Cox. 7 1/2" x 5 1/2", pp. 568, with 109 illustrations, some of them coloured. Price: 20s.

teriology must include, there is little scope for originality, but while in the main following conventional lines in the treatment of these aspects, "Bigger" occupies its own special and unique niche, created by the author's gift of clarity of expression, and by the simplicity and detail with which he describes essential laboratory procedures. With the passage of time, these naturally tend to be taken for granted by experienced workers, but, as Professor Bigger so sympathetically realizes, they may present bewildering problems to the student when first being initiated into the mysteries of bacteriology. The very emphasis placed on these points reveals such practical wisdom and patience, and long experience of teaching, that it also, however, provides interesting and refreshing reading for bacteriologists of higher status, particularly those responsible for ensuring that inexperienced juniors will not be sources of potential danger to themselves and their associates, and at the same time that they will quickly learn to produce useful and reliable work, by reason of technical competence and knowledgeable selection of material for diagnostic examination; it is a great comfort to be able to "delegate" part of such a task by advising close study by the novice of this useful handbook. One has only to think, for example, of the over-thick, over-stained films of, say, sputum, which form so many initial efforts, to appreciate the wisdom of Professor Bigger's guidance on this and similar seemingly elementary, but constantly recurring, problems.

Consideration of general or theoretical aspects of bacteriological problems are necessarily condensed in this book, and sometimes therefore appear dogmatic, but this seems inevitable, and perhaps even advisable, in view of the avowed objects of the author.

Chemotherapy, particularly in relation to penicillin, is dealt with in some detail in a special chapter present for the first time in this edition, and as would be expected, clear descriptions are given of technical methods found useful in the laboratory control of chemotherapy.

Another new chapter gives concise summaries of our knowledge of the viruses and rickettsiae known to be associated with human disease. In this field, Professor Bigger cannot use to the full his special skill, the description of practical procedures of diagnostic value, as these are not yet generally available in forms in which they can be used in "clinical" laboratories, in respect of diseases of viral origin.

This edition, as did its predecessors, largely omits reference to chronological sequence of events in the history of bacteriological advances, and also, like them, lacks a bibliography. In persisting with these omissions, the author is probably firmly holding to his conviction that these features are less essential to the purpose of his book than maximum compactness and devotion of all possible space to the practical aspect of the subject. With this, he deals in so able a manner that this handbook still deserves the widespread popularity it has enjoyed throughout the years since it first appeared in 1925.

THE ARTHROPATHIES.

A book entitled "The Arthropathies", by Alfred A. De Lorimier, M.D., is a second edition (1949),¹ the previous edition having been published in 1943. It is a handy little work and is well arranged, the illustrations being on the right-hand page with a concise description of the conditions illustrated facing it. This is a good arrangement for quick reference.

The present work has been improved greatly and the soft particulate appearances are described and illustrated as well as the bone appearances. Points of clinical and laboratory importance have also been added. Many new and illuminating descriptions and illustrations have been included and altogether the work shows a distinct and important advance on the previous edition.

The author states that a high quality skiagram may be described as containing the maximum of detail with optimum of contrast, the optimum of radiographic density and the minimum of distortion. Skiagrams must also display the surrounding soft tissues as much as possible. It is recommended that non-screen film technique be used rather than the intensifying screen in the study of the extremities. The tube-film distance should never be less than 30 inches, while 40 inches should be the minimum in the examination of the trunk. Higher kilovoltages are recommended than

are usual in practice. This is particularly of value in Potter-Bucky work.

The author lays down a method of routine analysis in the study of joints which should include the following aspects: the soft tissue, regional bones, articular cortex, articular cartilage, synovial membrane and joint capsule, the joint space. All these conditions must be based on tangible evidence and not on a "picture memory" of some past case. Part of the work deals with the peripheral joints covering developmental malformations and osteochondritis. The next group includes changes concerned with mechanical stresses, for example, osteoarthritis, and the third group affections associated with protein reactions, toxins and bacterial invasion.

Part II covers the joints of the spine and Part III the temporomandibular joint. This is a very satisfying arrangement. It is surprising that so many conditions have been adequately covered in so small a space and that such excellent illustrations have been gathered to cover these multitudinous pathological lesions.

One of the interesting sections is that dealing with peritendinitis. This includes those cases of irregular calcification about the shoulder and other joints. These conditions appear particularly about the insertions of the supraspinatus, infraspinatus and *teres major*. Excellent skiagrams are included showing the appearances seen in the various tendons.

Another excellent description with appropriate illustrations is given of the appearances seen in neuropathic conditions such as infantile paralysis and various anemic conditions. The various arthropathic conditions are described fully and well illustrated.

Altogether the book is very useful and every radiologist will find great help in his efforts to evaluate the various conditions occurring in diseased conditions of the joints.

IRON METABOLISM.

"IRON METABOLISM AND ITS CLINICAL SIGNIFICANCE"—a monument to the systematic research of its authors, Vannotti and Delachaux—was originally published in German in 1942. The present edition,¹ enriched by observations of radio-active isotopes in the study of iron metabolism and by further clinical experience, embodies many revisions of the original text and is a notable contribution to knowledge of the activities of iron in health and in disease.

Early chapters are devoted to the normal metabolism and to the biological significance of iron. In these are stressed: its indispensability as a constituent of the cell, cooperating in the formation and growth of the organism; its essentiality as an agent in total respiration, not only as an oxygen carrier in haemoglobin form, but as a biocatalyst in tissue oxidation and reduction processes; and its catalytic synergism with various hormones and vitamins.

Within the circulating blood, four individual iron fractions are described. These are not claimed as definite biological units, but are readily definable by physico-chemical methods, and, in that they show characteristic fluctuations in different pathological conditions, they have a diagnostic significance and shed some light on the underlying pathology.

Thus, in iron deficiency anemias, the serum iron fractions (as opposed to the haemoglobin iron) will necessarily be abnormally low, and it is suggested that the adynamia of these conditions and the oft associated fragility of finger nails and painful rhagades at the corners of the mouth are partly an expression of tissue iron deficiency rather than solely an outcome of lowered haemoglobin content—a conclusion based on the observation that these conditions may subside under treatment before erythropoiesis has approached normality.

A low serum iron in hypothyroidism would appear to be a sequence of the diminished total respiration (tissue and pulmonary) characteristic of low basal metabolism which, in reducing the need for iron, is a regulating factor in its absorption. In addition to this, the availability of iron for absorption will be reduced by the associated hypochlorhydria. It is therefore logical to assume that iron *per se* will make little contribution to the correction of associated anemia.

On the other hand many forms of hypochromic anemia showing a normal or even a raised serum iron content are presumably not iron deficiency anemias, and may be refractory to therapeutic iron. In such cases, aetiological conditions

¹"The Arthropathies: A Handbook of Roentgen Diagnosis", by Alfred A. De Lorimier, M.D.; Second Edition; 1949. Chicago: The Year Book Publishers, Incorporated. 7 1/2" x 5 1/2", pp. 338, with 157 illustrations. Price: \$7.00.

¹"Iron Metabolism and Its Clinical Significance", by A. Vannotti, M.D., and A. Delachaux, M.D.; translated by Erwin Pulay, M.D.; 1949. London: Frederick Muller, Limited. 9 1/2" x 6", pp. 270. Price: 32s.

to be sought are many and diverse—among others, a haemolytic factor; diets deficient in pyrrole bodies from which the porphyrin ring of haemoglobin may be synthesized; or even metastatic replacement of bone marrow from neoplasm.

The relation of the reticulo-endothelial system to iron metabolism is the subject of an interesting chapter. In this, the authors maintain that haemoglobin formation depends on the functional cooperation of the reticulum of the organs of blood decomposition which liberate and prepare the iron for further blood formation with the reticulum of the bone marrow which retains and stores the iron and, at an appropriate time, delivers it to the erythroblast.

In the anaemia of lead poisoning it is stated that lead, held in the reticulum of the bone marrow, prevents the delivery of iron to the erythroblast, thus inhibiting the formation of haemoglobin, but that it has no observable effect on the development of cytochromes of non-blood-forming tissues.

It is assumed, with some experimental justification, that iron stored in the reticulum may, in its capacity as an oxidizing catalyst, participate in the destruction of toxic bacteriological products.

Referring to the liver as an organ of excretion—one of its manifold roles in the metabolism of iron—it is observed that the excretion of biologically exhausted iron in the bile affords a means of reconditioning it in the intestine for reabsorption in a more useful form, thus completing a double entero-hepatic circuit.

An arresting proposition, which relates to some extent the blood changes occurring at great altitudes with the phenomena of *icterus neonatorum*, merits comment. The icterus is regarded as the product of an essential destruction of excessive haemoglobin which, necessary for oxygen saturation of a fetus developing in an oxygen-poor medium, is no longer needed when the fetus transfers to an oxygen-rich atmosphere. It is regarded as a benign and even purposive process, in that it affords a means of translating the haemoglobin iron to other forms needed for growth and for reserves.

With increasing knowledge of the complex metabolic activities of iron, its relations to total body respiration, and its functional collaboration with many vitamins and hormones, the authors predict that it will be prescribed more freely and rationally with such synergists as thyroloid, liver extracts, certain vitamins and perhaps bile salts.

The book ends with a short chapter on clinical indications and practical considerations in iron therapy. An extensive bibliography is appended.

For the clinician, divorced in medical practice from easy familiarity with biochemical entities and laboratory method, this book is by no means easy reading, but the effort affords ample compensation in the illumination it sheds on the whole field of rational iron therapy.

PROGRESS IN NEUROLOGY AND PSYCHIATRY.

THE annual review of "Progress in Neurology and Psychiatry", Volume IV, 1949, edited by E. A. Spiegel, M.D., maintains the high standard set by its three predecessors.¹ There are six members on the editorial board and seventy contributors, so that, as may be expected, there is a distinct variation in the quality of the numerous submissions which have, however, been skilfully brought together by the editor. A book such as this suffers from the fact that haste of compilation is essential, with the inevitable result that insufficient time is allowed for adequate critical evaluation of the material examined. Little in the literature has been missed. This volume should consequently be looked upon as a collection of data, the references to which are provided for consultation by the reader. In the 600 pages there are 34 reviews: five in the basic sciences of neuropathology, neurophysiology and pharmacology of the nervous system, nine in neurology, seven in neurosurgery and thirteen in psychiatric subjects. There is a comprehensive coverage of neuropsychiatric endeavour in 1948 from basic science to broad social aspects of psychiatry, including even such subjects as occupational therapy, rehabilitation and reeducation. This volume is essential for all post-graduate workers in the neuropsychiatric field and should be a work of reference in every medical library; beyond this, its field is limited, since summaries are scanty and a specialized perspective is demanded if the reader is to appreciate their significance.

¹ "Progress in Neurology and Psychiatry: An Annual Review", edited by E. A. Spiegel, M.D.; Volume IV; 1949. New York: Grune and Stratton. 9" x 5", pp. 612. Price: \$10.00.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The 1949 Year Book of Orthopedics and Traumatic Surgery (October, 1948-November, 1949)", edited by Edward L. Compere, M.D., F.A.C.S.; 1950. Chicago: The Year Book Publishers, Incorporated. 7" x 5", pp. 464, with 238 illustrations. Price: \$5.00.

A survey of recent literature.

"Modern Treatment Year Book, 1950: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Sir Cecil Wakeley, K.B.E., C.B., D.Sc., P.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S.; 1950. London: Baillière, Tindall and Cox. 8½" x 5½", pp. 388, with 23 illustrations. Price: 17s. 6d.

Current views on treatment.

"The 1949 Year Book of Neurology, Psychiatry and Neurosurgery (December, 1948-October, 1949)", Neurology—edited by Roland P. Mackay, M.D.; Psychiatry—edited by Nolan D. C. Lewis, M.D.; Neurosurgery—edited by Percival Bailey, M.D.; 1950. Chicago: The Year Book Publishers, Incorporated. 7" x 5", pp. 668, with 113 illustrations. Price: \$5.00.

A survey of recent literature.

"Practical Statistics in Health and Medical Work", by Ruth Rice Puffer, Dr.P.H., with a foreword by Hugo Muench, M.D.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 8" x 5", pp. 252, with a few illustrations. Price: \$3.75.

Gives some of the general principles of the analysis of statistical data and their practical use.

"The Sociology of the Patient: A Textbook for Nurses", by Earl Lomon Loos, Ph.D.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 8" x 5½", pp. 280, with a few illustrations. Price: \$3.00.

Intended to instil in the student nurse an understanding of her patient as a person.

"Methods in Medical Research", Editor-in-Chief, Julius H. Comroe, Junior; Volume II; 1950. Chicago: The Year Book Publishers, Incorporated. 8½" x 5½", pp. 377, with a few illustrations. Price: \$6.50.

Deals with methods of study of bacterial viruses, pulmonary function tests and assay of hormonal secretions.

"Training for Childbirth: A Program of Natural Childbirth with Rooming-In", by Herbert Thoms, M.D.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 8" x 5½", pp. 132, with a few illustrations. Price: \$3.00.

An account of the programme of training for childbirth at the Grace-New Haven Community Hospital.

"Practical Procedures in Clinical Medicine", by R. I. S. Bayliss, M.A., M.D. (Cambridge), M.R.C.P.; 1950. London: J. and A. Churchill, Limited. 8½" x 6", pp. 545, with 62 illustrations. Price: 25s.

Describes practical procedures commonly used in investigation and treatment, being primarily for senior students, house physicians, registrars and general practitioners.

"A Short Textbook of Radiotherapy for Technicians and Students, With a Supplementary Chapter for the Dermatologist", by J. Walter, M.A., B.M. (Oxford), M.R.C.P. (London), D.M.R.E. (Cambridge), and H. Miller, M.A., Ph.D. (Cambridge), F.Inst.P., with a foreword by J. L. A. Grout, M.C., F.R.C.S. (Edinburgh), F.F.R., D.M.R.E.; 1950. London: J. and A. Churchill, Limited. 8½" x 6", pp. 456, with 199 illustrations. Price: 28s.

Designed primarily for student radiographers preparing for examinations.

The Medical Journal of Australia

SATURDAY, JUNE 17, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

EMOTIONAL FACTORS IN OBESITY.

PERSONALITY STUDIES are now suggesting, and in some instances forcing, recognition of factors other than those which can be expressed in mathematical terms. Perhaps it is truer to say that when we look for the explanation of some obvious aberrations from the physiological norm we must admit that there are features that are hard to assess. The growth of psychiatry has posed some questions which are not easy to answer. Recent contributions to medical literature indicate that the difficulties and discouragements of attempting to reduce the excess weight of the obese raise such questions, and suggest an altered approach, at least with some patients. Spencer Bayles, in a brief review of the current psychiatric views on obesity in relation to personality, states that in most obese patients no metabolic disorder can be demonstrated.¹ He admits, of course, that the role of constitution, endocrine disturbance and heredity must be given just emphasis, and to these factors we may add those of age and exogenous factors such as the decreascent changes often seen after cultivated athleticism. As in most metabolic conditions needing treatment, the causal factors are mixed, often inextricably. Bayles points out that a basal postulate of psychiatry is that human behaviour may be explained and understood in terms of various fundamental needs. In applying this to the study of obesity he says that the needs of the person must be understood as well as those of his body. The obese patient is fat chiefly because he takes more food than he requires, and physicians find that he may readily lose weight if his intake is reduced below the inflated level which he has allowed it to assume. Theoretically this is simple, but when actual control of the patient's diet passes out of the hands of the doctor or the dietitian into the hands of the patient himself, the result is often frank failure, or at best only a modified success. The psychiatrist asks why this should be. If it is admitted that the intake has been in excess of the subject's minimal requirement for the needs of his body, what other needs is he satisfying by eating more than he

should? Bayles refers to the social aspects of eating, its symbolic significance, and its earliest connexions with affection, and suggests that the tension of hunger is not the only tension relieved by the taking of food. Those forms of tension familiar in everyday life are indicative of needs unfulfilled, and Bayles remarks that if this discussion is relevant to obesity it should be possible to find obese patients who eat because they are denied other pleasures, because they suffer boredom, or because they find that food symbolizes needs they cannot satisfy, such as affection. He draws attention to the recent publication of numerous case reports in which this thesis is sustained.

Perhaps it is germane to this inquiry to see if the penetrating understanding of life possessed by great exponents of the arts supports the crude summing up of the proverb that: "Nobody loves a fat man." We naturally turn to Holy Writ and to Shakespeare. Fatness is occasionally cited in the Old Testament as a mark of spiritual attainment and reward, but more often the opposite. The wicked, encompassed by the chain of pride, and clothed with violence, have eyes which stand out with fat. The wicked, thick of form and covered with fatness, lightly esteem the Rock of their salvation. Could we imagine a more compelling cautionary tale than the slaying of the oppressor Eglon, king of Moab, whose abdominal fat even closed over the specially made dagger which dispatched him? Perhaps we cannot draw medical morals from this viewpoint, but we may at least suggest that in those days psychological problems were simpler if not refined, and we can hardly expect to find the element of pity in the semibarbaric outlines of a people's travail.

Shakespeare, who did not allow the psychologist to displace the dramatist, has remade Falstaff for his own purposes, but even from this vast rolling figure of comedy and lewdness he has distilled something of compassion. We admit that wine played its part as well as food, but he compelled affection from some, even though he may have had the tinsel and not the gem. Was he so sure that the new-made young king's rejection of him as "so surfeit-swelled, so old and so profane" was official rather than personal? Even the brief story of his passing gains some sadness and regret. Two great musicians have described this same fat man also. Verdi made him a figure of gay comedy, yet the old man's reminiscence of his long ago service as page to the Duke of Norfolk expresses regret without irony. Elgar moves us to pity at Falstaff's frustration when the king disowned him, and, characteristically subjective, depicts the old rascal in a minor key that makes us feel sorry for him.

So much for literature and music; we may be wiser from reflection and insight, but it is still our problem as doctors to melt some of that too solid flesh from the victims of uncomfortable obesity. Our present author does not suggest that all obese patients should be passed over to the psychiatrist, but he makes the sound suggestion that instead of collecting exact data as to the amount the patient has eaten, it would be more profitable to spend more time in determining concomitant factors of environment and emotional life. In this way he thinks the patient may be brought to comprehend how he is making a non-metabolic use of food and how other avenues of satisfaction may open through greater insight. Surely a social understanding of the patient's life in

¹The American Journal of the Medical Sciences, January, 1950.

relation to his food requirements should help him; it is not enough to hand him a Spartan diet list which he is certain to discard after the first few weeks of conscious virtue.

Current Comment.

RADIOACTIVE IODINE IN THE DIAGNOSIS OF THYROID DISEASE.

THYROID DISEASE has been in the forefront of medical interest for a long time now; perhaps few disorders have stimulated work in so many fields. One of the most important of these fields has been that of clinical diagnosis, which has never been seriously threatened by the laboratory, in spite of the great help given by several precise methods. Bengt Skanse, who has worked on the problem of diagnostic methods for thyroid disease, and has made contributions on the subject, has now published a monograph in which he reviews the literature and describes his own work carried out in Boston for over two years.¹ His text opens the account; it is that, although typical cases of thyrotoxicosis usually pose no problem of diagnosis, the disease may present itself in forms which may trouble even an experienced clinician to diagnose. Even cardinal signs may be missing, and the mimicry of a number of other pathological states is often disturbingly close. The author examines first the validity of the recognized confirmatory tests. In doing so and in his original investigations he has drawn upon 110 normal subjects and 385 patients suffering from a variety of diseases. He had paid particular attention to the value of the estimations of the basal metabolic rate and of the serum cholesterol content, and then to the various efforts that have been made to establish tests for estimation of the blood iodine content and the excretion rates for iodine and radioactive iodine. He agrees that the basal metabolic rate offers a determination of great value in the diagnosis of thyrotoxicosis, provided that the rate is raised and that no other reason for this exists, and assuming that the test is carried out by experienced investigators. Surely clinicians will agree with his stricture that the test is of least value in the borderline cases in which help is most needed. The serum cholesterol test also is only of relative value. Much less useful than the basal metabolic rate determination, it resembles it in one particular, that serial determinations are of confirmatory value. Blood iodine estimations are unfortunately complex procedures which cannot be quickly and simply performed, and Skanse states that it is likely that some of the discrepant results in the literature are due to technical difficulties. The serum protein-bound iodine seems to parallel the level of the circulating thyroid hormone most accurately, and therefore to be the best of this group. An iodine tolerance test has not made a great deal of headway as yet. Iodine excretion tests were vitiated somewhat by the need for a large dose of iodine, which tended to obscure the avidity of the thyroid gland for the element, but the introduction of radioactive isotopes has opened a new field in this direction, for the urinary excretion rate may be much more sensitively measured, and the actual collection of iodine by the gland may be directly observed. It is along these lines that Skanse has made his observations.

The sections of the monograph dealing with methods are very interesting, and illustrate how much exact physical and mathematical knowledge is summoned to the aid of the modern researcher in these problems. The test substance used was I^{131} , with a half-life of eight days, being thus eminently suitable for clinical experiment. The radioactive iodine was standardized in absolute units, and its absolute disintegration rate was determined. The dose used in most of the experiments was 100 microcuries of I^{131} , but it was thought wise not to exceed the dose of 10 microcuries for the controls. To certain other patients

much larger doses were given prior to the removal of the thyroid, so that the excised glands could be studied by radioautography. It was therefore ascertained if the size of the dose affected the excretion in the urine; no influence on urinary excretion could be found. Care was also taken to insure that the small amount of carried iodine with the tracer substance did not invalidate a renal excretion test. Details of the apparatus cannot be dealt with here. The author claims to have established that a urinary excretion test for radioactive iodine is reliable and accurate and of practical value in the diagnosis of thyrotoxicosis. He has standardized the test on the excretion in the urine of I^{131} over two twenty-four hour periods after the oral administration of 100 microgrammes of inactive iodine "labelled" with 100 microgrammes of I^{131} . The results have been compared with the clinical diagnosis, most carefully determined, the basal metabolic rate, the serum protein-bound iodine, and the serum cholesterol. There has been no evidence of any untoward radiation effect of the doses employed. Of the 385 patients examined, 172 showed no signs of disturbance of thyroid function; 187 were in a hyperthyroid state, and 26 in a hypothyroid state. A large proportion of these patients showed atypical and confusing signs. In normal subjects $65.9 \pm 0.7\%$ of the test dose was excreted in the urine in forty-eight hours, 90% of this being passed in the first twenty-four hours. Age made little difference. This is the mean value, and it was found that the normal range lay between 44% and 87.8%. The patients with thyroid disease showed a very low excretion of I^{131} in 98% of cases. Study of the results of the basal metabolic rate, the serum cholesterol and the serum protein-bound iodine showed that these tests when taken alone missed 22% of the thyrotoxic subjects, whereas only 2% would have been missed by the radioactive iodine excretion rate alone. In no instance did patients with euthyroid states show any abnormality in the results of the excretion test. Possible disturbing factors of the test are the presence of renal disease, which may lower the rate, and the administration of iodine or desiccated thyroid, which may raise it. Moderately severe congestive cardiac failure did not invalidate the results. Skanse concludes that this test, when performed with proper safeguards, will reliably exclude non-thyrotoxic diseases, and is of particular value in thyrotoxicosis when the diagnosis is obscure, especially when the basal metabolic rate does not help. In fact he places it far above the estimation of the basal metabolic rate as a diagnostic aid, and its simpler technique, once the laboratory procedures and safeguards have been standardized, makes it more useful than the determination of the serum protein-bound iodine. The monograph will be of interest to all who are concerned with the advances of physical science in thyroid disease.

THE TREATMENT OF ACNE VULGARIS.

EVERY general practitioner has his quota of patients with *acne vulgaris*. Though he may or may not agree in entirety with the following statement, it will probably interest him:

In areas where dermatologic consultation is not available there is an occasional tendency for the physician to tell the patient that the condition is of no consequence and will be outgrown. Such an attitude is not justified, since each pustule that develops is a potential source of scarring, both physical and mental. Topical therapy and simple advice can bring about rapid improvement and frequently effect a complete cure.

This is the view of Helen Dexter¹ as the result of a recent special investigation, and though her report presents little that is new, the simple practical regimen of treatment suggested warrants consideration. Certain points brought to light in the investigation indicate useful lines of approach when these patients are questioned. The most

¹ *Acta medica Scandinavica*, Supplement 235, to Volume 136.

¹ *The Journal of the American Medical Association*, March 11, 1950.

important single dietary factor involved was chocolate; little justification appeared for great dietetic restriction, and as a general rule it was limited to chocolate, the "cola" drinks and excessive use of nuts and heavily fried foods. Faulty cosmetic habits were failure to wash the skin properly, stimulation of sebaceous secretion with complexion brushes, and the use of greasy cosmetics; the topical treatment recommended was designed to produce a satisfactory cosmetic effect. Postural habits are important, as Dexter confirmed the findings of others that acne is most pronounced where the hand touches the skin; it is helpful to warn patients of this fact. Emotional tension appeared to be significant and psychotherapy was of value in certain cases. Other aggravating factors concerned were the administration of iodides and of testosterone, occupational contact with oil and grease, menstruation, shaving (the use of electrical razors was helpful in some cases), hot weather and sunbathing. The topical therapy used, with complete or partial success in all cases, consisted first of all in application of soap to the affected areas twice in succession each morning and night. The skin was moistened with water as hot as could be tolerated; then, without undue rubbing, lather was applied with a soft cloth, left on for thirty to sixty seconds (depending on the type of skin) and finally rinsed with hot water. After two such latherings, the patient rinsed with cold water. The hot water and soap were intended to remove sebum and the cold water to decrease secretion. For darker or oilier skins a commercial abrasive soap was used, for fairer complexions a conventional milled toilet soap. Immediately after the cold rinse and drying, the patients applied a topical medicament of 2% of resorcinol, 8% of sulphur and 11% of alcohol in a flesh-tinted, non-greasy base ("Acnomel"). For patients with a thin, fair skin one application a day, or even every other day, usually sufficed. This preparation decreased the sebaceous secretion and was adequately keratolytic and antiseptic for all except deep pustules, and its cosmetic effect allowed treatment by day as well as by night. Where required face powder was used over it without ill effects. Dexter emphasizes that it is not necessary to open small pustules; with extensive cellulitis wet dressings should precede the use of the topical medicament until the acute phase is past, and with deep nodulocystic lesions, surgical and X-ray therapy must be considered. Good results are claimed for this treatment, and its value was supported by control use of other forms of treatment on the other side of the face. Although it requires attention to detail it is relatively simple; and, if it is as effective as is claimed, it should, as Dexter suggests, encourage the general practitioner to begin treatment of acne in the early phases of the disease before permanent damage has occurred.

BLOOD FORMATION AND DESTRUCTION AT HIGH ALTITUDES.

It is, of course, well known that polycythæmia is produced in persons living at high altitudes, that tolerance and adaptability vary with the individual, and that the increase in the circulating red cells is due to increased blood formation. Little attention has been paid to the necessary pendant to blood formation, blood destruction. South America has contributed in the past to our knowledge of the subject, and now César F. Merino presents the results of some studies carried out in Lima, Peru.¹ He points out that previous inquiries into the blood destruction of high altitudes have chiefly related to observation of the serum bilirubin, and the excretion of urobilinogen in relation to the quantity of hæmoglobin has not been studied. Recent work on the hæmolytic index, the ratio of faecal urobilinogen to the circulating hæmoglobin, suggested that this might be a good approach to the problem. Accordingly, he studied three groups of persons: (a) those normally living at sea level, at Lima, and over a test

period of twenty days at Morococha, 4540 metres above sea level; (b) those normally living at Morococha, and for five weeks at Lima; and finally (c) two men showing signs of failure of adaptation to life at this raised elevation. The blood was examined in the usual ways, the hæmoglobin value being calculated from the blood volume, which was estimated by the brilliant vital red method. The total bilirubin was determined in the serum, and daily estimations were made of the excretion of urobilinogen in the faeces and the urine. Colorimetric methods were used for both of these tests. The groups studied were naturally not large, nine in the first group and six in the second, but the choice of professional subjects, who gave intelligent cooperation, and the thoroughness of the methods used enabled trustworthy conclusions to be drawn. The hæmolytic index referred to above has been found to vary from 10 to 20, an index over 20 pointing to an acceleration in the process of blood destruction, even though the figure for faecal urobilinogen did not exceed normal limits. This enables the index to be used in subjects with anæmia or polycythæmia. In the first group it was found that three weeks was an insufficient period for the blood of subjects taken from sea level to a high elevation to assume the same degree of polycythæmia found in persons living there permanently, but the usual great increase in the total circulating red cells took place. In four or five weeks after return to sea level healthy persons were found to present a normal blood count. Natives habitually dwelling at 4540 metres began to show a diminution in hæmoglobin value and red cells from the first day of arrival at sea level, but such diminution, though progressive, was slow. The two subjects of chronic altitude sickness showed similar symptoms, fatigue, irritability, dizziness, insomnia, headaches, nausea, and inability to sustain exertion. They had conspicuous cyanosis and congestion of exposed surfaces, but the blood pressure was not raised above average, and physical examination revealed nothing else remarkable. The degree of polycythæmia found was in excess of that usual in the permanent dwellers at the same elevation, and the blood plasma volume was notably lessened in proportion to the increased mass of red cells. This is a distinguishing feature of chronic altitude sickness, and differentiates it from *polycythæmia vera*.

The most interesting part of the author's studies is that concerning pigment metabolism, which sheds light on the subject of blood destruction. His conclusions, linking up the cycle of formation, life and disposal of red cells under the stimulus of anoxia, are as follows. Polycythæmia due to a low pressure environment is brought about in the first few hours by blood concentration, but after forty-eight hours increased hæmopoiesis, unlike the more extensive cellular changes of *polycythæmia vera*, takes place, affecting the red cells only. After more prolonged exposure to the anoxia of high altitudes the blood volume increases, owing to increase in the total red cells, but not in the amount of the plasma, which is usually diminished. The excretion of urobilinogen does not rise at first, but later, as the red cells are produced and circulated in greater numbers, so more urobilinogen appears in the faeces. These changes run parallel, thus causing no disturbance of the hæmolytic index. The bilirubin in the blood increases in amount at the same time, but this cannot be explained by greater production, and seems to depend in part on lessened excretion by the liver. Thus, the normal person living at great heights has a polycythæmia characterized by proportional increase in both blood formation and blood destruction. This is in sharp distinction from what happens when such a person suffers a failure of his power of adaptation. Then polycythæmia of excessive degree appears, and this is associated with blood destruction out of proportion even with the increased blood formation. Perhaps most of these findings are what might be expected, but they supplement an interesting chapter in our knowledge of the life history of the erythrocyte under the strain of insufficient oxygen. The study of the pigments of blood and related pigments is of importance today in a number of different conditions, and additions to knowledge are welcomed.

¹ *Blood*, January, 1950.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Chorioidal Sarcoma with Metastasis in the Opposite Orbit.

SEYMOUR PHILPS (*The British Journal of Ophthalmology*, December, 1949) reports the case of a female, aged fifty-eight years, who in 1938 had the right eye removed because of the presence of a chorioidal sarcoma, the diagnosis being confirmed by pathological examination. In 1948 the left eye was noted to be proptosed and a provisional diagnosis of retro-orbital tumour was made. At operation an encapsulated tumour was found and removed, and histological examination showed it to be an encapsulated malignant melanomatous metastasis arising from a primary growth in the chorioid of the right eye. After operation the corrected visual acuity in the left eye was $\frac{1}{60}$.

Miotics.

EDWIN B. DUNPHY (*American Journal of Ophthalmology*, March, 1949) divides miotics into two groups—those which act directly on the effector cell in the iris muscle and those which act passively by inactivating or destroying cholinesterase and thus permitting the unregulated production of acetylcholine which stimulates the effector cell to activity. Those in the first group are closely related to acetylcholine and comprise "Mechoyl", "Carcholin" and "Furmethide"; pilocarpine acts in a similar manner. The second group, which destroys cholinesterase and thus allows uncontrolled production of acetylcholine, comprises eserine, neostigmin, fluorophosphates and tetraethyl pyrophosphate. The author makes detailed observations on individual miotics. He states that most miotics have a profound effect on the blood-aqueous barrier and on the osmotic pressure of the aqueous. Their action on the intraocular pressure is a complex one and represents a struggle between antagonistic forces. The use of a vasoconstrictor drug, as "Neo-Synephrine" in 1% solution, along with the miotic, is suggested as a means of combating excessive vasodilatation and cyclotonia. This use of a vasoconstrictor should be confined to shallow-angle glaucoma and acute congestive attacks.

Bilateral Total Ophthalmoplegia with Pituitary Adenoma.

FRANK B. WALSH (*Archives of Ophthalmology*, November, 1949) reports two cases of sudden, bilateral, total ophthalmoplegia with pituitary adenoma. The first was in a known acromegalic, who suddenly experienced excruciating pains in the head and face followed by bilateral total ophthalmoplegia. There was no fifth nerve involvement, and the corneal sensitivity was intact. Operation was followed by complete recovery from ophthalmoplegia, recovery of vision, and restoration of the visual fields to normal. The second case had a similar onset in an apparently healthy man. In this case corneal sensitivity was intact. After operation the ophthalmoplegia disappeared, but the patient remained blind. The ophthalmoplegia

is explained by pressure on the cavernous sinuses, and the retention of corneal sensitivity by the fact that sensory nerves continue to function, being more resistant to pressure than motor nerves, so that ophthalmoplegia may be complete before sensory loss occurs. In tumour of the cerebello-pontine angle there is corneal anaesthesia when other evidences of fifth nerve involvement are not demonstrable and there is incomplete facial palsy. It is believed that the association of incomplete palsies of the fifth and seventh nerves accounts for loss of the reflex, since each of these nerves forms part of the reflex arc.

Aureomycin in Ophthalmology.

STEWART DUKE-ELDER *et alii* (*The British Journal of Ophthalmology*, January, 1950) report the use of aureomycin in ophthalmology. For systemic administration the drug was given orally, 500 to 750 milligrammes every six hours up to a total of 20 grammes. For local application the borate salt was used. No serious toxic effects were noticed. The most striking results were obtained in eight cases of trachoma, all patients being treated by local application, one drop every four hours. All responded to treatment. Of the others treated, a patient with blepharitis due to a penicillin-resistant staphylococcus, one with conjunctivitis due to *Haemophilus influenzae* and one with inclusion conjunctivitis responded to treatment. Patients with chronic iridocyclitis, dendritic ulceration, superficial punctate keratitis, *herpes ophthalmicus* and sympathetic ophthalmia were treated without benefit.

Retinal Dysplasia.

ALGERNON B. REESE AND FREDERICK C. BLODI (*American Journal of Ophthalmology*, January, 1950) designate as "leukokoria" those conditions of infancy which produce a white reflex in the pupillary area as a result of opaque tissue at the back of the lens. They believe that in congenital "leukokoria" three entities can be recognized: retrolental fibroplasia, persistent hyperplastic vitreous and retinal dysplasia. Retinal dysplasia is a bilateral congenital malformation, manifesting itself at birth in full-term infants in association with cerebral agenesis and congenital anomalies elsewhere in the body. The eyes of babies with retinal dysplasia are usually microphthalmic, the condition is bilateral, and the lens, although clear at first, becomes cataractous. Pathologically the most characteristic feature of retinal dysplasia is malformation of retinal elements with the formation of true rosettes. The retina is detached and lies in folds against and enmeshed in vascularized connective tissue just behind the lens. There may be other anomalies, as coloboma of the uvea or optic nerve, or retrobulbar cyst.

Streptomycin and "Promizole" in Clinical Ocular Tuberculosis.

ALAN C. WOODS (*Archives of Ophthalmology*, December, 1949) presents a preliminary report on the treatment of twelve patients with ocular tuberculosis. He states that before the ocular condition was regarded as tuberculosis, the following criteria had to be fulfilled: the ocular disease must conform to a pattern commonly regarded as tuberculosis, the patient must show elsewhere in the body

evidences of preceding tuberculous infection, the cutaneous reactions to tuberculin must be in conformity with those for the patient's age and tuberculous status, and a thorough and exhaustive medical survey must reveal no other systemic disease or cause to which ocular inflammation can be logically attributed; finally all other forms of treatment must have failed, and the condition of the patient must be sufficiently desperate to warrant any risk that the treatment may entail. The patients treated with streptomycin and "Promizole" were grouped as follows: three had generalized uveitis and scleritis, four had sclero-keratitis and diffuse scleritis, four had severe exudative chorioiditis with involvement of the anterior segment of the eye, and two had Eales's disease. The full dosage of streptomycin varied between 26 and 168 grammes, given as 0.5 gramme every twelve hours, and the full dosage of "Promizole" varied from 98 to 2250 grammes, given as six grammes daily in divided doses. A response was observed after nine to twenty-one days. Toxic symptoms appeared in a large percentage of cases, but a therapeutic response was apparent before the appearance of toxic symptoms.

Cyclodialysis.

C. S. O'BRIEN AND JACK WEIR (*Archives of Ophthalmology*, November, 1949) are of the opinion that cyclodialysis is the operation of choice for any type of non-congestive glaucoma, for hydropthalmos and for secondary glaucoma following cataract extraction, and as a second or third operation after any unsuccessful previous surgical attempt to lower tension. They describe in detail the operation as performed by them and stress the importance of stripping almost half the attachment of the ciliary body. In an analysis of the results in 100 cases 79 were considered successful, the tension being 22 milligrammes of mercury or less and no loss of field occurring for at least one year.

Cranio-Facial Dysostosis (Crouzon's Disease).

MARSHALL M. PARKS AND FRANK D. COSTENBADER (*American Journal of Ophthalmology*, January, 1950) review the syndrome of cranio-facial dysostosis and report three cases. They state that it is a syndrome of cranio-facial deformity resulting from premature synostosis of the sutures of the base of the skull and of the face. The cause may be a true developmental anomaly of germinal origin, the fundamental defect being in the mesoderm which goes to make up the bones of the base and sides of the skull, or the defect may be in the germ plasm, manifesting itself by premature synostosis; the site of the congenital defect is the interstitial mesenchyme that normally separates the skull bones in their suture lines. The premature synostosis results in a skull that is unable to accommodate a normal and rapidly growing brain. The intracranial pressure is increased, but may be compensated by bulging of the bones; non-synostosed bones may be separated. The anterior fontanelle may be found to remain beyond the first year of age. The intracranial pressure depresses the fossae and increases the vertical diameter of the cranial vault. If intracranial hypertension is not adequately compensated, papilloedema or secondary

optic atrophy may occur. The premature synostosis of the zygomatic, maxillary, alisphenoid, temporal pterygoid, and palatine bones results in hypoplasia of the bones of the face and orbit, high arching of the palate and wide separation of the orbits with lateral direction. The peculiarly shaped and hypoplastic orbit forces the orbital contents forward, producing exophthalmos. The destructive cranio-facial deformity is conspicuous at birth. Sustained, decompensated, intracranial hypertension may provoke headaches or convulsions, or result in mental and visual impairment. If intracranial tension can remain compensated until the age of eight years, the prognosis is good for vision and mental state.

The Action of Eserine after the Use of Atropine.

C. R. S. JACKSON (*The British Journal of Ophthalmology*, March, 1950) reviews the literature on the action of eserine after the use of atropine and is able to show that eserine can affect mydriasis produced by atropine. He records the effect of eserine on the atropinized eyes of eight human subjects. In all cases miosis was produced with either 1.0% or 0.5% eserine solution. He points out that a great deal of temporary visual disability results from the use of 0.5% eserine solution three times a day patients would be enabled to resume work considerably earlier than would otherwise be possible.

OTO-RHINO-LARYNGOLOGY.

Tympanosympathetic Anaesthesia for Tinnitus Aurium and Secondary Otagia.

B. C. TROWBRIDGE (*Archives of Otolaryngology*, August, 1949) states that intrinsic tinnitus is a symptom of neural involvement occurring in disorders of the middle and the internal ear. The tympanic plexus, which constitutes the neural elements of the middle ear, is connected with the cochlear neuromechanisms of the internal ear through the *ramus vestibuli* and the *ramus cochlearis*. The tympanic plexus arises from the sensory fibres of the trigeminal, sympathetic and glossopharyngeal nerves. Through the connexions of these nerves tinnitus aurium may be produced by pathological changes in the middle ear and, in neighbouring structures, such as teeth, pharynx, tongue, sphenoidal sinus and nose, and Eustachian tube. The impulses arising may be manifested either by tinnitus or by aural pain. Circulatory disturbances of the internal carotid artery are responsible for tinnitus of a pulsating or throbbing kind. Another likely source of vascular tinnitus is the middle meningeal artery. The "peach glow" or "flamingo red" hue of the promontory in otosclerosis indicates hyperemia of the mucosa of this region, in which the tympanic plexus is located, and may thus be related to the tinnitus, which is often an initial symptom. Ethylmorphine hydrochloride, when injected into the tympanum, has proved to be effective in the treatment of tinnitus and secondary otalgia because of its combined anaesthetic and analgesic qualities. The drug also acts as a vasodilator and lymphagogue stimu-

lating the vascular and lymphatic circulation. Immediately after injection the entire drum membrane becomes congested. The patient experiences little discomfort owing to the analgesic action of the drug. The author describes the technique of injection, which is carried out under local cocaine anaesthesia. He states that the method is surprisingly free from uncomfortable or disturbing reactions. The treatment consists of successive instillations of the drug at intervals of four days. Tinnitus and otalgia may be abolished in the majority of cases after five treatments. In other cases a second series of injections may be required after an interval of two months. When there is not complete loss of tinnitus the symptom may be decreased to the point at which it is no longer a source of annoyance. Improvement of hearing is often observed.

Arytenoidectomy for Bilateral Abductor Paralysis of the Vocal Cords.

WILLIAM C. THORNELL (*Archives of Otolaryngology*, November, 1949) states that various surgical procedures have been devised for the correction of bilateral abductor paralysis of the vocal cords. Kelly described a procedure in which arytenoidectomy was carried out through a small window in the thyrocartilage. Others have suggested modifications of this procedure along with various additional steps aimed at fixation of the posterior end of the vocal cord in a widely abducted position. The author makes an intralaryngeal approach using the Lynch suspension laryngoscope under combined local and intravenous anaesthesia. An incision is made over the superior surface of the arytenoid cartilage and extended antero-laterally into the aryepiglottic fold. Submucous dissection of the arytenoid cartilage is then carried out. After removal of the cartilage a curved electrocautery point is deeply inserted through the incision and along the course of the thyro-arytenoid muscle beneath the vocal cord. Further lateral fixation of the cord is thus attempted by means of the contracture resulting from the electrocautery. Haemorrhage is minimal and readily controlled with the cautery. Tracheotomy is performed prior to the operation. Laryngeal oedema is pronounced for the first three or four days and usually has subsided by the tenth to the fourteenth day. The cannula is removed twenty to thirty days after operation. Three cases are described in which dyspnoea and noisy respiration were adequately relieved.

Modified Radical Mastoidectomy.

S. H. BARON (*Archives of Otolaryngology*, March, 1949) states that the matrix of cholesteatoma should be preserved as a contribution towards healing. The cholesteatoma may be considered as an epithelial cyst. Danger from the presence of this cyst is removed once it is adequately opened and the factor of pressure eradicated. The matrix of squamous epithelium may then be used to assist in epithelialization of the cavity. Numerous surgeons have recorded experiences to disprove the necessity of removing the cholesteatoma matrix, including Dundas-Grant, Kopetzky, Shambaugh, junior, Seibenmann, Almour and others. The author has been utilizing the matrix of the cholesteatoma in most cases, including both radical and modified

radical operations, since 1933, and attests to the soundness of the procedure. He states that surgical intervention in cases of chronic suppurative otitis media is indicated particularly in the type of case with a marginal perforation, this type being considered as dangerous in contrast with the non-dangerous type with a central perforation. Since the dangerous otorrhea is in the ear that has a marginal perforation, and the latter more often involves the *pars flaccida* or the upper or posterior part of the *pars tensa*, it is logical to expect the modified radical mastoidectomy to be indicated more often than the radical. This has been the author's experience in practice. He uses a modification of the Lempert endaural incision, and preserves the membranous part of the external auditory canal, as in Lempert's fenestration procedure, for use as a flap for covering portions of the operation cavity. The opening of the mastoid process and epitympanum is performed with electrically driven burrs, care being taken not to disturb the ossicles. If cholesteatoma is present the matrix should not be disturbed. The membranous portion of the external auditory canal finally remains as a tube of skin, which may then be cut and fashioned so that the inner segment is used to line the cavity of the mastoid bowl, and the outer portion is turned upwards to cover the roof of the meato-mastoidal operation cavity. The flaps are held in place with packings of a lace-mesh surgical dressing, which remain in place for five or six days.

Radium for Hypertrophic Lymphoid Tissue in the Naso-Pharynx.

CHARLES M. DOW (*Archives of Otolaryngology*, October, 1949) states that during the past ten years radium has been found to be useful in destroying recurrent lymphoid tissue in the nasopharynx. However, the dose of radiation has not been definitely established. The results are compared in two short series of cases (numbering 41 and 33 respectively) in which a "Monel" metal 50-milligramme radium sulphate applicator was placed for eight and a half minutes and twelve minutes respectively on each side of the naso-pharynx. The treatment was employed for a variety of conditions in both adults and children; these included recurrent adenoids, deafness, recurrent and chronic otorrhea, lateral pharyngeal bands and granular pharyngitis, and post-nasal discharge. A single applicator was used, one side being treated immediately after the other. Three treatments were given at intervals of two weeks. Nineteen patients were treated for hearing defects. Seven of these were given the eight and a half minute dosages, and in four hearing was restored to a practical level; lesser improvement was obtained in two cases, and in one there was no change. Of twelve patients in whom the time of application was twelve minutes, the hearing was restored to a practical level in all. Similar results were obtained in cases of recurrent central adenoid tissue. In cases of lateral pharyngeal bands and granular pharyngitis and of post-nasal discharge, there was very little difference in the effects obtained with the two periods of dosage. It is concluded, from comparison of the two series of cases, that the longer treatment removes lymphoid tissue safely and more effectively.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Royal Adelaide Hospital, Adelaide, on March 30, 1950, the President, Dr. C. O. F. RIEGER, in the chair. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Spontaneous Hydro-Pneumothorax.

DR. K. V. SANDERSON showed a patient who had had spontaneous hydro-pneumothorax. The patient, a single man, aged twenty-four years, had been admitted to the Repatriation General Hospital, Springbank, on December 29, 1949, with the provisional diagnosis of malaria. For some days he had had an alternate-day fever with headache and malaise. He had last been in the tropics four years before. When examined he was found to have a temperature of 100.2°F ., a pulse rate of 106 per minute, and a respiration rate of 18 per minute. His spleen was palpable one centimetre below the left costal margin and he was tender below the right costal margin. Examination of a blood film showed no malaria parasites. On the same night he developed pleurisy in the lower part of the chest on the right side and a cough with thick yellow sputum. His temperature the next morning was 103°F ., and his respiration rate 28 per minute. He was cyanosed and was obviously ill. Examination of the chest showed diminished movement of the right side with impaired percussion note and decreased breath sounds at the right lung base. A provisional diagnosis of right basal pneumonia was made, and he was treated with penicillin, 30,000 units every three hours. A white cell count revealed 19,100 leucocytes per cubic millimetre, of which 87% were polymorphonuclear cells, 3% lymphocytes, 9% monocytes, 3% eosinophile cells and 1% basophile cells. Culture of the sputum produced a light growth of both hemolytic and non-hemolytic streptococci. On the following day his symptoms were unchanged; examination now showed evidence of a patch of consolidation of the lung below the angle of the right scapula, and a pleural friction rub was heard in the right axilla. The spleen was not palpable. Dr. Colin Gurner made the following report on an X-ray picture of the chest taken later in the day: "A right pneumothorax with effusion showing a horizontal level to the fourth anterior rib. There may be some underlying lung condition in addition." During the next week the patient slowly improved. His pain left him and his cough decreased. The signs in his chest were those of pleural effusion with a pneumothorax above. A little pleural fluid was removed four days after the penicillin therapy had been started, and this contained 2700 leucocytes per cubic millimetre, predominantly polymorphonuclear cells. The fluid was sterile, and the results of guinea-pig inoculation and culture for *Mycobacterium tuberculosis* were negative. The result of the Mantoux test was negative.

The patient was then examined by Dr. Guy Lendon, who suggested that he had had a cystic condition of his lung which had become infected and ruptured into the pleural cavity. As the patient had lived in the country as a child, Dr. Lendon suggested that a Casoni test be performed, but owing to an oversight that was not done. At the end of the second week the patient became apyrexial and the penicillin therapy was stopped. The right pleural cavity was aspirated and 44 ounces of clear yellow fluid were removed. X-ray films taken after aspiration showed that a small amount of fluid remained with evidence of many loculated areas due to adhesions. There was compression of the lower and middle lobes, but no evidence of collapse. The patient's health improved over the next three weeks; he had little cough and no sputum. However, the fluid reaccumulated and the lung showed no signs of reexpansion despite the fact that further tapplings were performed. On February 3, 1950, Dr. J. G. Sleeman and Dr. H. D. Sutherland examined him in consultation and recorded the following opinion: "The clinical course and X-ray films suggest a rupture of a weakened lung cyst or bleb, with an infective condition of the pleural cavity rendered abortive by antibiotic therapy. It is recommended that exploratory thoracotomy be carried out to allow inspection of the lung surfaces and to facilitate reexpansion of the lung." Dr. Alan Penington also examined the patient and gave a similar opinion. On February 8, 1950, Dr. Sutherland performed thoracotomy through the bed of the seventh rib. The pleural cavity was cleared of fibrinous and fluid debris. A hydatid cyst was located and removed with a considerable amount of its adventitious capsule, and

the raw area of the lung oversewn with chromicized catgut. Decortication of the visceral pleura was performed. The wound was closed after an apical and a basal under-water seal drainage tube had been placed in position. The macroscopic findings were that the pleural cavity contained air and fluid divided into loculi by fibrinous masses. The lung occupied only about half the hemithorax. A ruptured hydatid cyst was situated in the posterior basal fringe of the lower lobe of the right lung. Its adventitious capsule was about three inches in diameter, and this in turn had ruptured, causing a broncho-pleural fistula and air leak on the diaphragmatic surface. After decortication the three lobes expanded fully. Microscopic examination of the cyst wall showed the typical laminated structure of the wall of a hydatid cyst. No hooklets or scolices were found in the encysted pleural material. Convalescence was somewhat prolonged, because of the persistence of some degree of air leak, but with the under-water seal drainage, supplemented by suction, and with breathing exercises, the lung expanded fully, and the patient was discharged from hospital on March 20, 1950, with an excellent result. During the period of convalescence a hydatid complement-fixation test was carried out and the result found to be negative.

In discussing the course of the illness, Dr. Sanderson said that it was probable that a small leak into a bronchus had been established, allowing the entry of pathogenic organisms. That focus of suppuration had caused the feverish symptoms prior to admission, but provided few signs by which it could be located in the lung. After the patient's admission to hospital some of the pus from the hydatid cyst had begun to drain into the bronchial tree, possibly giving rise to some degree of bronchopneumonia in the adjacent lung tissue. At the same time the pleura had become involved, with resultant pleurisy. The cyst then ruptured through the pleura, discharging its infective contents and establishing a broncho-pleural fistula. The infection was controlled by antibiotic therapy, and by the time fluid was aspirated it was sterile and contained few cells. Dr. Sanderson pointed out that the occurrence of a pyopneumothorax was one of the less common complications of a hydatid cyst of the lung, and in the case under discussion there seemed little evidence to suggest the diagnosis before operation, except that the patient had lived in the country. The miniature X-ray picture of the chest taken on his discharge from the service had been normal, and there had been no eosinophilia in the blood at the time of his admission to hospital. The case was instructive in that it showed the necessity of always considering hydatid disease in the differential diagnosis of a cystic condition of the lung.

In conclusion, Dr. Sanderson said that he desired to thank the Chairman of the Repatriation Commission for permission to report the case.

DR. DARCY SUTHERLAND said that the operation was performed in the case under discussion primarily to restore the function of the lung by decortication, but also with the hope that the cause of the spontaneous hydro-pneumothorax could be found and treated accordingly. He said that he thought before the operation that the most likely diagnosis was a ruptured emphysematous bulla with pleural infection which had been almost controlled by chemotherapy. The pre-operative X-ray film, showing loculated collections of fluid and thickened fibrin over the surface of the lung, indicated that without decortication the function of the lung on that side would be very restricted. At operation the ruptured hydatid cyst was found in the costo-phrenic angle, the cyst having been situated in the extreme fringe of the lung. The fact that the cyst was unsupported by lung itself had probably resulted in its rupture into the pleura rather than into a bronchus as was more common. The post-operative course was uneventful with the exception of an air leak into the pleural cavity, coming either from the damaged area of lung or from one of the small pleural tears always resulting from decortication. Usually those tears closed within a few days, but in the case under consideration under-water seal drainage was necessary for almost four weeks before complete expansion resulted.

Achalasia of the Cardia.

DR. A. R. SOUTHWOOD discussed two cases of achalasia at the cardiac orifice occurring in two brothers. The first of the patients had come to the Adelaide Children's Hospital in September, 1936, at the age of six and a half years, on account of persistent vomiting of three months' duration; he was vomiting daily, and sometimes after each meal. The appendix had been removed six weeks before his admission to hospital. At first the condition subsided with rest and administration of sedatives, but it recurred, and he remained in hospital until early in December, 1936. He was readmitted

on several occasions. He was in hospital from early April, 1938, until his death in October, 1939, at the age of ten years. The diagnosis of achalasia of the cardia had been confirmed by X-ray examination and swallowing of a barium bolus. Treatment was effective: the mercury loaded rubber bougie number 32 was used, and the boy was able to pass it himself. He had numerous attacks of bronchospasm with asthma. The X-ray report of May 4, 1937, revealed that after the barium was swallowed the oesophagus was dilated and cardiospasm was marked by only small amounts passing through. After a large piece of cake and further barium were swallowed, some bronchospasm developed and the oesophagus showed excessive dilatation with active peristalsis. Adrenaline was then injected and the bronchospasm was relieved, but there was no effect on the cardiospasm. The illness was mostly afebrile, but in May, 1939, irregular pyrexia began and continued till his death in October. In May, 1939, he developed pneumonia. X-ray examination showed a large area of opacity in the right mid-zone of the right lung. He died from pneumonia and perforation of the oesophagus.

The second patient had first come to the out-patient department at the Children's Hospital at the age of nine and a half years on account of mild weakness of the legs; slight adductor spasm was noted. In September, 1946, he was admitted to the Adelaide Children's Hospital. He had had measles in July, and since then had been eating poorly and was vomiting after meals. Achalasia of the cardia was diagnosed and confirmed by X-ray examination and swallowing of barium. The taking of a large drink of water after each meal was followed by relief from vomiting. X-ray examination in December, 1946, showed no delay at the cardia. The large bougie was passed easily. The boy left hospital on January 6, 1947. He was admitted to the Port Pirie Hospital on February 14, 1950, with signs of pneumonia affecting the mid-zone of the right lung. After three weeks of febrile illness he was transferred to the Verco Ward at the Royal Adelaide Hospital. During his three weeks in Verco Ward the pyrexia had continued with evening rises varying from 100° F. to 102° F. There had been a chronic cough with no sputum. He had had 12 million units of penicillin (one million units daily), and since March 26 had had a quarter of a gramme of streptomycin twice daily. X-ray examination of the chest had revealed a fairly extensive opacity suggestive of pneumonia in the upper lobe of the right lung with a similar but less extensive opacity in the mid-zone of the left lung and small patches of opacity apparently due to consolidation in each lower lobe. X-ray examination with the swallowing of a barium bolus revealed enormous dilatation of the oesophagus, the lower end tapering almost to a point with a smooth, rounded, lower contour. A well-marked area of opacity was present in the base of the upper lobe of the right lung and the oesophagus was appreciably deviated to the right. The relationship between the dilated oesophagus, its deviation to the right and the opacity in the right lung field was not obvious. The features of the oesophagus suggested cardiospasm, and that might cause kinking to the right.

DR. DARCY SUTHERLAND said that he had seen several Heller type operations whilst in England. Results had been almost universally satisfactory. However, he felt that as a cure rate of 70% to 80% was to be expected from hydrostatic dilatation through an oesophagoscope, Heller's operation should not be resorted to in the first instance. He also thought that permanent damage had probably occurred in the lung and doubted if that could be cured without resection.

Reconstruction of the Urethra.

DR. NOEL J. BONNIN showed a patient on whom operation had been performed for reconstruction of the urethra.

DR. G. H. BURNELL discussed the case and said that he thought that use of a suprapubic drain might be necessary for three weeks after operation.

Paralysis of the Larynx.

DR. R. M. GLYNN showed a patient with bilateral paralysis of the larynx treated by the Kelly operation. He stated that the paralysis had followed a thyroidectomy, and a tracheotomy had been performed soon afterwards, as the patient complained of considerable respiratory distress. The arytenoidectomy was performed about twelve months later. The patient was able to do her ordinary work, and was completely satisfied with the result.

Superior Vena Caval Obstruction.

DR. MALCOLM W. MILLER discussed a case of obstruction of the superior vena cava.

Secondary Carcinoma of the Lung.

DR. B. S. HANSON showed the X-ray films of a patient with secondary carcinoma of the lung.

DR. DARCY SUTHERLAND said that he had not seen a secondary deposit from a carcinoma of the breast producing one large lesion in the lung. He would be interested to know if anyone else could remember such a case. He thought that in view of the rarity of such a deposit of carcinoma from the breast, the fact that the lesion could be seen bronchoscopically, together with its microscopic appearance and its response to X-ray therapy, indicated that it was more likely to be an undifferentiated squamous carcinoma of bronchogenic origin.

Desmoid Tumour of the Abdominal Wall.

DR. G. H. BURNELL discussed a case of desmoid tumour of the abdominal wall. He said that it was a local malignant fibroma that did not metastasize. It might occur secondarily to trauma—in the case under discussion, cholecystectomy. It usually occurred in the rectus muscle. Removal in the case under consideration appeared to have been successful.

DR. C. O. F. RIEGER, from the chair, said that 80% of cases of the type under discussion occurred in women.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on April 12, 1950, at the Children's Hospital, Carlton, Victoria.

Fibrocystic Disease of the Pancreas.

DR. CHARLOTTE ANDERSON stated that West, Wilson and Eyles (writing in the *American Journal of Diseases of Children* of September, 1946) had described the amino-acid absorption curve as a reliable diagnostic test in fibrocystic disease of the pancreas. In this disease there was deficiency in protein digestion due to the absence of pancreatic secretion containing trypsin. Proteins were not split to the amino-acid and peptide stage, and consequently were not absorbed. Thus the normal postprandial rise in blood amino-acid nitrogen level, which had been demonstrated by a number of workers, would not occur in cases of fibrocystic disease of the pancreas. Working on that theory, West *et alii* had studied the rise in blood amino-acid nitrogen content over a five-hour period following the giving of a protein meal, and in a very limited series of controls and cases of fibrocystic disease, had plotted two types of curve—the normal with a quick rise and fall, and the fibrocystic which had a "flat" form, with a slow and small rise throughout the whole five-hour period.

The ultimate diagnosis of fibrocystic disease of the pancreas had rested, up to the present, on the absence of trypsin in the duodenal contents, which were obtained by intubation. That was a technically difficult, time-consuming and sometimes harmful procedure. The blood amino-acid absorption test seemed to be a much simpler procedure, if it could be proven to be reliable. Therefore they had tried to study a comprehensive series of normal children, as well as patients with fibrocystic disease, and patients for whom the differential diagnosis rested between fibrocystic disease and other conditions characterized by steatorrhea and chronic or subacute chest infection. The test was the same in detail as that used by West *et alii*. The rise in blood amino nitrogen level was studied during a five-hour period following a protein meal. Gelatin, 1.74 grammes per kilogram of body weight, in liquid form and flavoured with sugar and orange juice, was given to the child, 0.2 millilitre of blood was collected after an eight to ten hour fast, and then at intervals of half, one and a quarter, two and a half and five hours after the meal. The amino-acid nitrogen content was estimated by the colorimetric method of Krauel *et alii*, as described in *The Journal of Laboratory and Clinical Medicine* of February, 1944.

In the group of normal control children 45 subjects in all were studied, being divided into the following age groups: birth to six months, six to twelve months, one to three years, three to six years, and over six years. They were children previously well and active, entering hospital for some minor surgical procedure, such as circumcision or hernia operation, and the test was always performed before operation. Others amongst the older children were recovering from some mild illness, such as "U.R.T.I.", tonsillitis *et cetera*. A curve derived from the following average values (in milligrammes

per centum) was obtained from all normal patients: fasting, 3.0; half an hour after meal, 4.4; one and a quarter hours after meal, 5.6; two and a half hours after meal, 5.5; five hours after meal, 4.1. The maximum rise occurred about one and a quarter hours after the meal, and then a fall followed towards the fasting level at the end of five hours. However, there was considerable individual variation in the results. The fasting level varied between 2.5 and 7.5 milligrammes per centum, with the majority between 3.0 and 5.0 milligrammes per centum. No significant difference occurred between the average values obtained in each age group, but there was much individual variation as could be seen in the curves derived from the two following sets of values (expressed in milligrammes per centum): fasting, 5.0 and 4.5; half an hour after meal, 7.0 and 4.1; one and a quarter hours after meal, 7.3 and 4.8; two and a half hours after meal, 11.8 and 5.6; five hours after meal, 7.3 and 4.6. The second case was the only one of 45 in which the rise above fasting level was not more than 1.5 milligrammes per centum.

Dr. Anderson said that 16 cases of fibrocystic disease of the pancreas had been studied, and the typical curve obtained in most cases was that termed a "flat" curve. The average curve of all of the cases was as follows, based on the following values (in milligrammes per centum), all except one case having been checked by duodenal intubation, and five by post-mortem examination: fasting, 4.4; half an hour after meal, 5.0; one and a quarter hours after meal, 5.1; two and a half hours after meal, 5.5; five hours after meal, 5.4. However, in three cases, curves not fitting into this pattern were obtained, being derived from the following sets of values (in milligrammes per centum): fasting, 4.8, 4.3 and 6.0; half an hour after meal, 4.6, 4.7 and 9.4; one and a quarter hours after meal, 4.9, 5.8 and 6.9; two and a half hours after meal, 6.0, 8.8 and 11.1; five hours after meal, 6.4, 6.7 and 7.6. No adequate explanation could be made for the variants, but the fact that they did occur was important, showing that the test was not always reliable, and all cases of fibrocystic disease did not give a uniformly "flat" curve.

The first case of the disease studied was that of a boy, aged nine years, on whom it had been impossible to pass a duodenal tube owing to persistent cough; he died a few months later and post-mortem examination established a diagnosis of fibrocystic disease. The second case was that of a baby, aged six months, the only finding in which was the passage of fatty stools since birth. The child was well nourished and had made excellent progress up to the age of two years. However, duodenal intubation revealed absence of trypsin, and the curve obtained on two occasions was of a flatter type. The third case was that of a baby who had typical features of the disease (chronic cough, failure to thrive, steatorrhoea, *Staphylococcus aureus* in sputum), but duodenal intubation revealed the presence of a normal amount of trypsin. Post-mortem examination, however, confirmed the diagnosis of fibrocystic disease of the pancreas.

The third series of cases studied included, firstly, two cases of coeliac disease, in both of which curves were obtained within the normal range. In both also normal duodenal tryptic activity was present. Secondly, five cases of steatorrhoea were studied, including three of undetermined aetiology, and two associated with rectal stricture. In all of these, curves of a normal type and normal tryptic activity were found. Thirdly, there were six cases of subacute chest infection associated at some stage in the illness with steatorrhoea. In all normal tryptic activity was present in the duodenal fluid, and in all except one an amino acid curve was obtained within normal limits. In one case, however, a slowly rising curve occurred with a total rise of only 1.2 milligrammes per centum at the end of five hours. However, there were other facts in the case which were against the diagnosis, such as absence of *Staphylococcus aureus* from the sputum, atypical X-ray findings, and continued improvement without dietary change.

In summing up, Dr. Anderson said that the amino-acid absorption curve was a useful screening test in the investigation of patients suspected of suffering from fibrocystic disease of the pancreas, and should be carried out before duodenal intubation, as it was a simple, harmless test and would make duodenal intubation unnecessary in certain cases. It was not a reliable diagnostic test of fibrocystic disease, as some patients with the disease had a curve which fell into the range of curves from normal children. If the results of the test were interpreted in conjunction with the clinical pattern, and with the radiological and bacteriological evidence of the disease, then the test provided useful supporting evidence in establishing a diagnosis of fibrocystic disease.

Dr. HOWARD WILLIAMS said that fibrocystic disease might be defined as a general disorder of mucus-secreting cells

of the body, which was manifested clinically, firstly by the signs and symptoms of pancreatic achylia, secondly by a diffuse infection of the respiratory tract by *Staphylococcus aureus*, and thirdly by defective nutrition and growth. There was a strong hereditary basis for the disease. The three groups of signs and symptoms were often present in the initial phases of the disease; in the later or terminal phases they were almost without exception present. However, at the time of the initial examination a considerable number of patients had signs and symptoms referable to only one of the groups, for example, the signs and symptoms of defective pancreatic digestion or those of chronic pulmonary infection. Nevertheless, if the signs and symptoms were predominantly pulmonary, then a careful clinical search often revealed some evidence of disordered digestion, and vice versa.

The pulmonary structure of patients with fibrocystic disease was normal, with perhaps the exception of the mucus-secreting cells. Such changes as occurred were the result of infection. The essential pathological changes in the lungs were the result of infection of the bronchial tree causing bronchitis and bronchiolitis. The infecting organism was almost invariably the *Staphylococcus aureus*. The thick, viscid exudate from the bronchitis resulted sooner or later in one or a number of the following pathological changes: obstructive emphysema, collapse, bronchopneumonia, and abscess formation and bronchiectasis. Although the changes were usually widespread and diffuse, they might be localized to one lobe or one portion of the lung. Commonly a number of the changes were present at any one time. Thus one patient might have phenomena of collapse, emphysema and "pneumonitis". The diffuse bronchiolitis, if severe and extensive, caused obstructive emphysema which reduced very considerably the vital capacity. Dyspnoea was therefore a common symptom, and anaemia and cyanosis occurred if the obstructive emphysema was severe.

The physical signs of the pathological changes in the lungs were mainly those dependent on emphysema and bronchitis. In the advanced disease the thoracic cavity was distended by the voluminous lungs, the sternum was thrust forwards and the diaphragm downwards, and the increased respiratory effort often resulted in reaction of the soft chest wall to form a Harrison's sulcus. Areas which were normally dull to percussion, such as the liver, the heart and the inferior part of the thorax posteriorly, became resonant. Rales were heard where exudate was disturbed by breathing. The following radiological signs were seen: (i) an increase in the broncho-vascular markings of the lungs from the vascular engorgement associated with the bronchitis; (ii) increased hilar shadows from the swollen hyperaemic lymph glands and increased vascularity of bronchi in the lung root; (iii) mottling in the lung fields from patchy areas of bronchopneumonia and multiple lobular areas of collapse; (iv) pulmonary collapse of a segmental or lobar distribution; (v) emphysema, of either segmental or lobar distribution, or diffuse generalized emphysema from diffuse obstructive bronchiolitis.

A considerable number of the patients presented with pulmonary symptoms which so dominated the clinical picture that a primary lung lesion was suspected as the cause of the illness. A more detailed clinical history and examination would show that many of the patients had abnormal stools. Nevertheless, with some patients the sole manifestation of the disease was, in the initial stages, referable to the pulmonary system. Three case histories of patients with proven fibrocystic disease illustrated the points made. The first patient, a healthy full-term infant, of healthy parents, had been born after a normal labour. At the age of three weeks she had contracted a "cold", and since then had had a persistent cough and later coughed up small pellets of gelatinous greenish mucus. At the age of five months the child had more than doubled her birth weight and was sturdy and well developed. Her stools and appetite were normal. Clinical and radiological examination of the child at five months of age disclosed a collapsed upper lobe of the right lung. Culture of the sputum grew *Streptococcus viridans*. A Mantoux test with 1 in 1000 old tuberculin produced a negative result. Over a period of four months, the child's symptoms persisted, and gradually cavitation appeared in the upper lobe of the right lung and at the same time her general health deteriorated. At seven months of age and again at eight months the child developed widespread bronchitis and bronchopneumonia with gross obstructive emphysema, death occurring in the later attack when she was aged nine months. Only in the last two weeks prior to death was any abnormality noted in the stools; they then became bulky and offensive. Post-mortem examination disclosed generalized purulent bronchitis, bronchopneumonia, extensive emphysema and a collapsed bronchiectatic upper lobe of the right lung and a pancreas with classical evidence

of fibrocystic disease. The patient was regarded as having a primary lung lesion for many months, as during that time there were no clinical features suggestive of any disorder of digestion or nutrition. It was only in the last few weeks of illness that the underlying basis of the pulmonary lesion was suspected.

The second patient, the fourth child of healthy parents, had been brought to the Children's Hospital for examination as the baby had been in contact with her grandfather, who was found to have pulmonary tuberculosis. She was a well-developed baby, aged three months and weighing twelve pounds. Clinical examination revealed no abnormality, the Mantoux test yielded a negative result, but X-ray examination of the chest disclosed collapse of the lower lobe of the left lung. A diagnosis of the pathological cause of the pulmonary collapse was not made. The child made good progress until the age of five months, when she developed a cough, which was dry and irritating and caused her to vomit. On examination at that time she was a well-nourished baby, with stools normal on clinical examination; crepitations were audible over the posterior part of the left side of the chest and in the left axillary region. Radiological examination of the chest showed increased vascular markings with considerable emphysema and collapse in the lower lobe of the left lung. Although the stools appeared normal, microscopic examination showed the presence of excess fat, culture of a pharyngeal swab grew *Staphylococcus aureus*, the amino-acid curve was "flat", and no trypsin was present in the duodenal juice. The baby was in hospital for eleven weeks, and during that time she had a constant temperature, with persistent clinical and X-ray signs in her chest, and a constant irritating cough. Her stools later became rather bulky and offensive. The *staphylococcus* infecting her respiratory tract was found to be resistant to both penicillin and streptomycin. The child showed some improvement on treatment with these drugs, however, and was discharged to her home. She had made slow progress and at the time of the meeting was eleven months of age; but she still had her cough and signs of infection in the chest. The diagnosis of fibrocystic disease was not suspected as being the underlying cause for the pulmonary collapse that was accidentally discovered in this child at the age of three months. The reason was that there were no other clinical features in any way suggesting the possibility of such an aetiology.

The third patient was the first child of healthy parents and had made satisfactory progress until she was over two years of age. In her second year of life she had had several colds which cleared up satisfactorily. When aged two years and three months she developed an irritating, dry, spasmodic cough, which later caused vomiting. Her illness was naturally attributed to pertussis by her family doctor as the clinical features seemed typically those of pertussis. It was only when the cough and chest signs failed to clear after some weeks that he suspected an infective complication of the illness. Examination at the Children's Hospital at that stage, ten weeks after the initial illness began, revealed a wasted little girl with extensive emphysema of her lungs and crepitations audible over the chest; X-ray examination showed extensive mottling and emphysema over the entire lung fields. Sputum culture grew *Staphylococcus aureus* and *Bacillus coli communis*, and her stools continued excess fat. She made temporary improvement with penicillin therapy. At this stage her stools became bulky and offensive, and a diagnosis of fibrocystic disease was made. She went home for a number of months, but at the age of two years and nine months the chest infection flared up, and she died shortly afterwards. Post-mortem examination showed a typical fibrocystic pancreas, extensive purulent bronchitis, bronchopneumonia and emphysema, and early bronchiectasis. The illness in the initial stages had presented features typically like those of pertussis. There were no clinical features at that stage that could have suggested that fibrocystic disease was the underlying aetiological factor.

Dr. Williams pointed out that the three patients with fibrocystic disease had all presented with a pulmonary lesion with no clinical evidence of any underlying abnormality of digestive function or growth. The children were normal in growth and development, and their stools were normal. Only after the lesion in the lung had been present for some time were there features suggesting fibrocystic disease as being the underlying pathological condition. Those features were abnormal stools with excess fat, excess bulk and an offensive odour, together with some general wasting and failure to grow. Dr. Williams summed up by saying that the initial clinical manifestations of fibrocystic disease might be solely pulmonary, and other clinical features of the disease might not appear for many months. Fibrocystic disease should be considered as a possible aetiological basis in patients with a subacute or chronic chest lesion,

which was characterized by emphysema, bronchitis and collapse, especially if the lesion was widespread and involved the upper lobe as frequently as the lower lobe.

Dr. KATE CAMPBELL asked what part the *succus entericus* played in protein digestion in patients with fibrocystic disease.

Dr. Anderson replied that it must have a definite role as some children thrived very well. The exact function was not known at present. West had claimed that bacterial digestion of the protein might be responsible for the late rise in the blood amino-acid curve in patients with fibrocystic disease, but that opinion was hypothetical and lacked any proof.

Dr. J. COLEBATCH asked whether the fasting level of blood amino nitrogen was lower in patients with a poor nutritional state as compared with those in a good state of nutrition, and also if the level was lower in the very early age group.

Dr. Anderson replied that there seemed to be no definite relationship between the nutritional state and the fasting level of blood amino nitrogen. The fasting level was higher in older children than in babies, but insufficient numbers of children had been investigated to make the statement statistically valid.

Dr. Colebatch also asked if any patients with fibrocystic disease had been found to have a normal trypsin level in the duodenal contents and then at a later stage it had disappeared.

Dr. Anderson stated that she had not seen such a patient.

Dr. STANLEY WILLIAMS asked if extensive pulmonary changes in fibrocystic disease were capable of extensive resolution under treatment.

Dr. Howard Williams replied that they were, but it was impossible to predict the change in any one individual patient.

Correspondence.

CONJOINED TWINS.

SIR: In your annotation on "Conjoined Twins" (May 20, 1950), your commentator suggests that the term *dicephalus dibrachius*, found in "Blakiston's New Gould Medical Dictionary", might be preferable to *thoracopagus tribrachius dipus*, as used by R. J. Riddell in his description of a monster in the same number of the journal. I am inclined to agree with the commentator that a shorter, more easily understood term might have advantages over the somewhat cumbersome, though more explicit, usual teratological designation, but surely the correct form would be, again on the authority of the dictionary, *dicephalus tribrachius*.

Yours, etc.,

H. F. BETTINGER.

Department of Pathology.

The Women's Hospital,

Melbourne.

May 24, 1950.

["Blakiston's New Gould Medical Dictionary" (page 1258) gives the characteristics of *dicephalus dibrachius* as "two heads; partly double thorax; two arms", and those of *dicephalus tribrachius* as "two heads; partly double thorax; more or less complete third, median arm". It is a matter of opinion whether or not "a stump representing a third arm on the postero-superior aspect between the two necks" may reasonably be classed as a "more or less complete" arm. Neither term is exactly right for what is an intermediate form; either should surely be acceptable.—EDITOR.]

A MORE REALISTIC VIEW OF TUBERCULOSIS.

SIR: On reading Dr. B. Short's first letter on this subject (THE MEDICAL JOURNAL OF AUSTRALIA, February 18, 1950, page 249), I was prepared to let pass some of his statements on tuberculosis, in view of the value of his letter in drawing attention to the dangers of "over-investigation" and of treating the X ray instead of the patient. However, Dr. Short's second letter (THE MEDICAL JOURNAL OF AUSTRALIA, May 27, 1950, page 713) shows that his experience of the disease differs greatly from mine.

In his first letter (*vide supra*), Dr. Short says that "the mortality must be extremely low". H. O. Lancaster (1950) gives 4719 deaths in Australia in the five-year period 1941-1945, and for the same period he states that, for the age group of fifteen to forty-four years, tuberculosis caused

16.28% of all deaths. I do not consider this to be a low mortality.

In the same letter, he appears to believe that mass radiography is going to reveal all the primary, as well as the post-primary, lesions, and so result in half the population receiving sanatorium treatment. This has not happened in those countries which have widespread mass radiography. Ontario had only found it necessary to provide 3.7 beds per annual death (Roche, 1944). The reason, of course, is that most primary, and many post-primary, lesions are subminimal.

In both his letters, Dr. Short gives the impression that he believes that symptomless cases are mostly minimal, or even negligible. This has not been my experience. Although a single case proves nothing, may I offset the impression created by Dr. Short's cases with the following experience, which, in lesser degree, is frequent.

An accountant, middle-aged, active, and feeling in good health, brought up about one pint of blood while rehearsing with his repertory society. An X ray the next morning showed extensive cavitation of both lungs, with infiltration of all zones. This finding was confirmed *post mortem* three days later.

This man, and hundreds like him, may have been saved by mass radiography. And the thousands who contracted the disease from them may have been saved their morbidity. At the other end of the scale, subminimal lesions can cause symptoms.

All of which shows that definite answers cannot be given to most of Dr. Short's questions. A decision on activity can only be given by a chest physician, on the available facts. It is an act of judgement, and therefore will be wrong occasionally. But the error occurs both ways.

As a trainee psychiatrist, I would say that the answer to his question "Does this change [to activity] conveniently occur when the lesion becomes recognizable radiographically as a shadow . . ." is no.

I agree with Dr. Short that there should be research in connexion with any mass radiography campaign. But I also agree with Dr. Bruce White (1950) and others, when they state that, with our present knowledge, tuberculosis could be eliminated in from thirty to fifty years by widespread radiography and appropriate treatment.

Mass radiography is obviously the first step in the fight against the disease. The governments will not provide sanatorium beds for treatment until we find the cases to put in them, and the cases and the public demand facilities for treatment.

In conjunction with radiography, there must be education of the public, in the manner advocated by Dr. Bruce White (1950).

I believe that the fight against tuberculosis will be slow until it is treated as an infectious disease, that is, by isolation of infective cases.

In conclusion, may I ask Dr. Short what better planned form of attack he suggests, and wherein lies the "semi-politically inspired 'bone-pointing'?"

Yours, etc.,

V. L. MATCHETT.

Mental Hospital,
Beechworth,
Victoria.
May 31, 1950.

References.

- Lancaster, H. O. (1950), "Tuberculosis Mortality in Australia, 1908 to 1945", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume I, page 655.
Roche, Hilary (1944), "Some Observations on Tuberculosis Control", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume II, page 52.
White, B. (1950), *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume I, page 386.

THE BRISBANE CONGRESS: AN APPRECIATION.

SIR: I have just returned from the seventh session of the Australasian Medical Congress (British Medical Association) in Brisbane. My dominant impression of my week's stay in Brisbane—a week of glorious sunny days and mild nights and a scientific and social calendar full to overflowing—is of the marvellous hospitality of our colleagues and of the people of Queensland. In fact, I have been so touched that I feel impelled to write this open letter of thanks and ask you if you think it would be appropriate to print it.

The gracious patronage of the Governor, who came in person to two of our meetings and, I understand, entertained our representatives at Government House; the hospitality of the Premier, who arranged for us to see the sights; the

great interest of the Press and the cheerful greetings of shop and hotel people were much commented upon and appreciated by the visitors. In my "sponsor", one of the Brisbane doctors, I discovered a kindred spirit and a most friendly fellow. He and his family were more than kind to me: to meet them has been a very great pleasure indeed—that is putting it mildly—and I hope that our friendship may last. Apart from my sponsor there were several other doctors and their wives who had me out to their homes or took me to parties: through their kindness I met a number of people after my own heart as well as some contemporaries whom I had not seen since our university days. I do not know that I learned very much from the scientific sessions and I contributed very little, but I think that the cobwebs were brushed out of a good many of the medical departments of my mind.

I do not want my name under this letter if you print it: from the remarks of nearly every doctor I spoke to, I gather that they feel as I do and thus, possibly, would not be averse from sharing its authorship, so I sign myself just

Yours, etc.,

"CONGRESSMAN."

Sydney,
June 5, 1950.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Professor Francis G. Blake, M.D., M.A., D.Sc., F.A.C.P., Professor of Medicine in the Yale University School of Medicine, New Haven, Connecticut, United States of America, will give the following four lectures in Sydney:

Friday, June 30, 1950: "Present Status of Antibiotic Therapy, with Particular Reference to Aureomycin, 'Chloromycetin' and Terramycin."

Monday, July 3, 1950: "Hepatic Diseases."

Wednesday, July 5, 1950: "The Treatment of Meningitis."

Friday, July 7, 1950: "The Use of ACTH and 'Cortisone'."

All lectures will be given in the Stawell Memorial Hall, 145 Macquarie Street, Sydney, at 8.15 p.m.; admission is by ticket only. Tickets may be obtained on application to the Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, the enrolment fee of £1 is. being enclosed.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 30, of May 25, 1950.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Gordon Dunlop Kirkness is appointed Surgeon Lieutenant (D) (for Short Service), dated 15th April, 1950.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Grant of Honorary Rank.—John Kempson Maddox is granted the honorary rank of Surgeon Commander, dated 12th April, 1950.

Transfer to Retired List.—Surgeon Lieutenant-Commander Neil William George Macintosh is transferred to the Retired List, dated 11th April, 1950.

Resignation.—The resignation of John Kempson Maddox of his appointment as Surgeon Commander is accepted, dated 11th April, 1950.

Royal Australian Naval Volunteer Reserve.

Colin Graham Alderman is appointed Surgeon Lieutenant, with seniority in rank of 24th May, 1944, dated 31st July, 1949.

Surgeon Lieutenants Ronald Munro Ford and John Hamilton Stace are promoted to the rank of Surgeon Lieutenant-Commander, dated 10th March, 1950.

Obituary.

GUSTAVE HEUZE HOGG.

We are indebted to Dr. C. Craig for the following appreciation of the late Dr. Gustave Heuze Hogg.

The death of Dr. Gustave Heuze Hogg removes one who did a great deal for the advancement of medicine in Launceston. Dr. Hogg was born in 1869 at Finnisterre in Brittany. He was educated at the Launceston Church Grammar School, and after winning the Tasmanian Scholarship at the age of eighteen years, he went to Edinburgh to study medicine. He was Gilchrist Scholar later at the University of London, when he took a classics degree. He took his M.D. degree at Edinburgh, and later studied obstetrics at the Rotunda, Dublin. He returned to Australia in 1891 and practised at Cooma, New South Wales. In 1893 he returned to Launceston and practised in partnership with the late Dr. George Maddox. His lifelong interest was ophthalmology, and in 1909 he returned to Europe for special study in this specialty, working in Paris and Vienna.

With the late Dr. G. E. Clemons and the late Mr. John Gunning, he founded the Queen Victoria Maternity Hospital at Launceston.

In 1925, after a long absence, the honorary medical officers returned to the Launceston General Hospital. There had been a quarrel with the Government and for several years the hospital was closed to outside medical men. Dr. Hogg was very prominent in the negotiations leading up to the return, and himself took the position of honorary ophthalmologist, which he held until his retirement in 1935. He published many papers on ophthalmology. He was a member of the Ophthalmological Society of the United Kingdom and a Fellow of the Royal Society of Medicine, London, as well as a life member of the British Medical Association, a past president of the Tasmanian Branch of the British Medical Association, and a Foundation Fellow of the Royal Australasian College of Surgeons. He was a member of the Australasian Medical Publishing Company, Limited, from 1913 to 1947.

Dr. Hogg's community interests were wide. He was a prominent Mason, was president of the Launceston Mechanics' Institute Library for years, founded the Saint Andrew's Caledonian Pipe Band, was a member of the Council of the University of Tasmania, was a member of the Board of the Church Grammar School for many years, and was its chairman for eight years.

Dr. Hogg was a tall man, of distinguished presence and manners. He was a very good speaker at clinical meetings and never failed to emphasize that medicine was an art and only partly a science. He was very fond of references to the classics and to the great French clinical teachers, such as Trousseau. He and the late Dr. G. E. Clemons were intimate friends, and Launceston owes much to them for their work at Launceston General Hospital and at the Queen Victoria Hospital. Dr. Hogg is survived by his widow, two sons and a daughter.

HUGH ALTON CHANDOS WALL.

We are indebted to Dr. Kevin Byrne for the following appreciation of the late Dr. Hugh Alton Chandos Wall.

Hugh Wall was a likeable type of man. Educated at Fort Street School, in 1915 he graduated from the University of Sydney with the degrees of M.B., Ch.M., and immediately left as one of "Kitchener's Hundred Doctors", recruited in the Dominions to proceed to the help of the Mother Country on active service abroad. Wall went with the British Expeditionary Force to Gallipoli, whence he was invalided from Lemnos to Australia. He was one of the few British officers to be given a farewell from all and sundry. In Sydney he was appointed resident medical officer to Sir Alexander MacCormick at Saint Vincent's Hospital; but having regained his health, he again felt the call to arms. He left Sydney as surgeon lieutenant in the destroyer flotilla, Royal Australian Navy, of which His Majesty's Australian Ship *Parramatta* was flagship, and which was based at Brindisi and swept the Adriatic for Austrian submarines. Although nominally in the *Parramatta*, Wall never

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 27, 1950.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory. ³	Australian Capital Territory.	Australia. ²
Ankylostomiasis
Anthrax
Beriberi
Bilharziasis
Cerebro-spinal Meningitis	2(2)	2(2)	1(1)	1	6
Cholera
Coastal Fever(a)
Dengue
Diarrhoea (Infantile)
Diphtheria	14(8)	4(2)	5(4)	1	3	27
Dysentery (Amoebic)
Dysentery (Bacillary)	..	3(2)	1(1)	4
Encephalitis Lethargica
Erysipelas
Filariasis
Helminthiasis
Hydatid	..	2	2
Influenza
Lead Poisoning
Leptosy
Malaria(b)	..	1(1)	1(1)
Measles	18(12)	2	20
Plague
Polymyositis	11(9)	2	..	15(11)	2(2)	30
Psittacosis
Puerperal Fever	1(1)	..	2(1)	3
Rubella(c)	1	1
Scarlet Fever	20(13)	21(8)	5(3)	8(2)	..	9(2)	..	3	66
Smallpox
Tetanus	..	1	1
Trachoma
Tuberculosis(d)	35(28)	22(19)	23(17)	5(4)	18(15)	5(4)	108
Typhoid Fever(e)
Typhus (Endemic)(f)	1	2(2)	1(1)	4
Undulant Fever	1(1)	1
Well's Disease(g)
Whooping Cough	1(1)	1
Yellow Fever

¹ The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946-1947. Figures in parentheses are those for the metropolitan area.

² Figures not available.

³ Figures incomplete owing to absence of returns from the Northern Territory.

⁴ Not notifiable.

(a) Includes Moxman and Sarina fevers. (b) Mainly relapses among servicemen infected overseas. (c) Notifiable disease in Queensland in females aged over fourteen years. (d) Includes all forms. (e) Includes enteric fever, paratyphoid fevers and other *Salmonella* infections. (f) Includes scrub, murine and tick typhus. (g) Includes leptospirosis, Well's and para-Well's disease.

knew in which ship he would sleep, as "press gang" officers from other ships would "shanghai" him onto their vessels to enjoy his delightful company and interesting conversation, which helped to while away the monotony of the two days' "sweep". A hobby of his was on long week-ends to travel with some fellow characters to north Italy, get access to the trenches, and enjoy the busman's holiday of watching the Italians fight. Possibly this may have engendered the rumour which spread that the Allies were short of soldiers as sailors were in the trenches. Hugh was very well and widely read, his strange choice of subject being mediæval French. In later life he settled in private practice on the north coast of New South Wales and did much for charity.

FRAMPTON GARNSEY MEADE.

We regret to announce the death of Dr. Frampton Garnsey Meade, which occurred on June 3, 1950, at Clayfield, Brisbane.

FRANK COUPER WOOSTER.

We regret to announce the death of Dr. Frank Couper Wooster, which occurred on June 5, 1950, at Rockhampton, Queensland.

Honours.

BIRTHDAY HONOURS.

The following are included among the Birthday Honours awarded by His Majesty the King.

Dr. Neil Hamilton Fairley, of London, has been created a Knight of the Most Excellent Order of the British Empire.

Dr. Wilberforce Stephen Newton, of Melbourne, has been created a Knight Bachelor.

Dr. William Keverall McIntyre, of Launceston, has been created a Commander of the Most Distinguished Order of Saint Michael and Saint George.

Dr. Hugh McIntyre Birch, of Adelaide, has been created a Commander of the Most Excellent Order of the British Empire.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Nash, Thomas Paul, M.B., 1948 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Nash, Stanley Allan, M.B., B.S., 1950 (Univ. Queensland), 44 South Parade, Campsie.

Corrigan, Raymond John, M.B., 1948 (Univ. Sydney), 22 Carabella Street, Kirribilli.

Toffer, Oswald Boaz, M.B., B.S., 1950 (Univ. Sydney), 61 Fitzgerald Street, Bondi Junction.

Notice.

AUSTRALIAN ORTHOPÆDIC ASSOCIATION PRIZE FOR 1950.

THE honorary secretary of the Australian Orthopaedic Association has asked us to announce that the Australian Orthopaedic Association Prize for 1950 has been awarded to Dr. Max Lake, Brisbane Hospital, Brisbane, for his essay on *ostetis deformans*.

AN APPEAL FOR JOURNALS.

THE New South Wales Red Cross Blood Transfusion Service is endeavouring to establish a medical library. Some numbers and indices of *The Lancet* (1946 onwards), *British Medical Journal* (1940 onwards) and *THE MEDICAL JOURNAL*

OF AUSTRALIA (1940 onwards) are missing from its files and are not obtainable from the publishers. The Red Cross Society would appreciate any help that medical practitioners may be able to give in developing this library. Particulars of the missing numbers will be supplied by the director of the Blood Transfusion Service, 374 George Street, Sydney (BW 1684).

Diary for the Month.

JUNE 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.

JUNE 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.

JUNE 21.—Western Australian Branch, B.M.A.: General Meeting.

JUNE 22.—New South Wales Branch, B.M.A.: Clinical Meeting.

JUNE 22.—Victorian Branch, B.M.A.: Executive Meeting.

JUNE 22.—South Australian Branch, B.M.A.: Clinical Meeting.

JUNE 23.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to *THE MEDICAL JOURNAL OF AUSTRALIA* alone, unless the contrary be stated.

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